

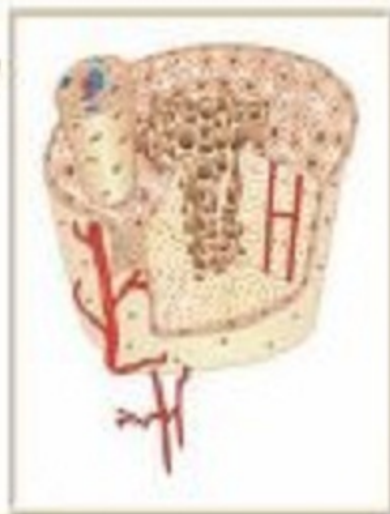
Textbook of Craniofacial Growth



Sridhar Premkumar

Foreword
KSGA Nasser

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Textbook of Craniofacial Growth

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Reader

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***Dedicated to
the memory of
my parents
Shri KP Sridhar and
Mrs Sreemathy Sridhar
and
to all my teachers
who have played a role in
my growth and development***

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Foreword

Science has developed phenomenally and likewise the field of orthodontics has also witnessed a phenomenal amount of development. It is believed that writing a textbook is an obligation of the expert academician towards students as well as to his colleagues in pursuit of continuing their education. An understanding of craniofacial growth is essential for all dental surgeons and specialists like orthodontists, pedodontists and maxillofacial surgeons. This textbook on craniofacial growth has been divided into 21 chapters, starting from development of bone and cartilage till the etiology of developmental and acquired deformities. It has been written in a lucid, fluent style which can be appreciated and understood by the average student as well as practitioner.

The importance of this book lies in the simplicity of its presentation which helps everyone understand the concept and principles of craniofacial growth. Every BDS undergraduate should understand the concept of orthodontics right from the basic level so that he or she can practice confidently as an individual. The design and presentation of this book shows the meticulous and tireless effort of the author. The book covers the craniofacial growth and its impact on orthodontics in detail. I am sure that this book will be of great help to dental students and also to academically oriented dental surgeons.

I wish the author, for all success in his future endeavors.

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Preface

There is growing emphasis on the quality of treatment results, particularly from the aspects of prevention, interception and correction of malocclusion. In spite of the level of knowledge acquired in their preclinical years, students are unable to recall and relate facts and problems related to their training. This problem is more specific in relation to the subject of craniofacial growth and development.

Craniofacial growth is a wonderful and fascinating phenomenon and understanding the fundamental aspects of dentofacial growth and development is vital for every clinician to effectively deal with the complex problem of abnormal skeletal growth of the jaws and dentoalveolar malocclusion. It is the author's intent that this book will be of value to those studying their undergraduate course in dentistry and more specifically, those who undergo specialization training in the fields of orthodontics, pedodontics and oral and maxillofacial surgery.

The writing of a book is beset with many responsibilities. These are serious when the first edition is written. The tide of many new thoughts ebb and flow through the years. The author must keep in mind the decision of including the subject matter relevant to the intended purpose. The justification of presenting another textbook on craniofacial growth to the shelves of dental literature is to be found in the presentation of subject matter and novelty of its presentation. Adequate care is taken to ensure that this textbook on craniofacial growth is complete in most of the aspects. Even development of emotional growth is included as a separate chapter. I sincerely hope that the book will be of immense use for both the faculty and students, as the book is conceived both as a teaching and a reference guide. It is hoped that this book together with future orthodontic research will provide the bridge to a new era in the holistic treatment of the orthodontic patients.

Please mail your feedback to dr_premsidhar@yahoo.co.in

Sridhar Premkumar

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Dr Ramya Julian, Dr Catharin Maney, Dr Sathish Kumar and Dr Srirengalakshmi deserve special thanks for their selfless and constant help in bringing out this book. Though many students have helped in different ways, a note of gratitude to Saurab Jain, Sushma R, Prateshta Rajeevi Arvind and Sumaiya Parveen.

No book can be better than its publisher. The author would like to thank the publisher Shri Jitendar P Vij of Jaypee Brothers Medical Publishers (P) Ltd, and also Mr Jayanandan and Mr KK Raman for their effective coordination.

Closer to his home, the author would like to acknowledge his son Sriram and daughter Srinidhi, his energy boosters, for their innocent love and affection. Finally, most fortunate authors have tolerant wives behind them. The author's wife has been one, having lost him for innumerable hours to the computer!

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Biology of Bone and Cartilage

CHAPTER OUTLINE

- **Classification of Bone**
- **Functions of Bone**
- **Gross Structure of Long Bone**
- **Types of Bone Tissue**
- **Molecular Structure of Bone**
 - The matrix of bone
 - Composition of bone
 - Collagens
 - Cells of bone
- **Skeletogenesis/Bone Formation**
 - Membranous ossification
 - Endochondral ossification
 - Initiation of calcification or Mineralization
- **Mechanisms of Bone Growth**
 - Chondral growth
 - Sutural growth
 - Periosteal growth
 - Remodeling
- **Cartilage**
 - Structure of cartilage
 - Lubrication mechanism of cartilage
 - Cartilage replacement mechanisms

Bone is essentially a highly vascular, living, constantly changing, mineralized connective tissue. Bone tissue is the structural material that gives the bones the strength they require to act as levers for muscles, to give form to the soft tissues of the body, and to provide protective cavities for the vital organs. However, bone tissue also serves as a mineral bank that can be drawn upon in times of need. Morphologically bone tissue appears to be under the control of bone cells. Its surfaces are enveloped by active and resting osteoblasts and osteoclasts, and it is permeated by an interconnected canalicular system in which osteocytes are found. Bone is a connective tissue distinguished by the fact that its

matrix is mineralized by calcium phosphate in the form of crystals very similar to hydroxyapatite. Bone is a tension adapted tissue and tension on the periosteum will result in the differentiation of osteoblasts from the periosteal cells and leads to formation of new bone. On the other hand, pressure on the periosteum will cause vascular occlusion and may produce bone resorption.

CLASSIFICATION OF BONE

Bones can be classified based on their position, shape, size, and structure.

Based on location, bones can be classified as follows:

- *Axial skeleton*—Bones of the skull, vertebral column, sternum, and ribs.
- *Appendicular skeleton*—Bones of the pectoral girdle, pelvis girdle, and limbs.
- *Acral skeleton*—Part of the appendicular skeleton, including bones of the hands and feet.

Based on shape, bones can be classified as follows:

- *Flat bone*—Bones of the skull, sternum, pelvis, and ribs.
- *Tubular bone*
- *Long tubular bone*, including bones of the limbs.
- *Short tubular bone*, including bones of the hands and feet, such as the phalanges, metacarpals, and metatarsals.
- *Irregular bone*—Bones of the face and vertebral column.
- *Sesamoid bone*—Bones that develop in specific tendons, the largest example of which is the patella.
- *Accessory bone or supernumerary bone*—Extra bones that develop in additional ossification centers or bones that failed to fuse with the main parts during

development (Accessory bones are common in the foot and may be mistaken for bone chips or fractures).

Based on size, bones can be classified as follows:

- *Long bone*—Tubular in shape, with a hollow shaft and 2 ends, including bones of the limbs.
- *Short bone*—Cuboidal in shape, located only in the foot (tarsal bones) and wrist (carpal bones).

FUNCTIONS OF BONE

Bones have the following main functions:

- *Protection*—Bones can serve to protect internal organs, such as the skull protecting the brain or the ribs protecting the heart and lungs.
- *Shape*—Bones provide a frame to keep the body supported.
- *Blood production*—The marrow, located within the medullary cavity of long bones and the interstices of cancellous bone, produces blood cells in a process called haematopoiesis.
- *Mineral storage*—Bones act as reservoir of minerals important for the body, most notably calcium and phosphorus.
- *Helps in Movement*—Bones, skeletal muscles, tendons, ligaments and joints function together to generate and transfer forces so that individual body parts or the whole body can be manipulated in three-dimensional space. The interaction between bone and muscle is studied in biomechanics.
- *Maintenance of Acid-base balance*—Bone buffers the blood against excessive pH changes by absorbing or releasing alkaline salts.
- *Detoxification*—Bone tissues can also store heavy metals and other foreign elements, removing them from the blood and reducing their effects on other tissues. These can later be gradually released for excretion.
- *Transduction of sound*—Bones are important in the mechanical aspect of hearing.

GROSS STRUCTURE OF LONG BONE

The gross structure of a long bone can be divided into several regions.

Epiphysis: In the long bones, the epiphysis is the region between the growth plate or growth plate scar and the

expanded end of bone, covered by articular cartilage. An epiphysis in a skeletally mature person consists of abundant trabecular bone and a thin shell of cortical bone. Although an epiphysis is present at each end of the long limb bones, it is found at only one end of the metacarpals (proximal first and distal second through the fifth metacarpals), metatarsals (proximal first and distal second through fifth metatarsals), phalanges (proximal ends), clavicles, and ribs.

The epiphysis is the location of secondary ossification centers during development. The structure of the epiphysis is more complex in bones that are fused from more than one part during development. Examples include the proximal and distal ends of the humerus, femur, and vertebrae. For instance, the proximal end of the humerus is developed from three separate ossification centers, which later coalesce to form a single epiphyseal mass. In the proximal humeral epiphysis, one of the centers forms the articular surface, and the other two become the greater and lesser tuberosities. Carpal bones, tarsal bones, and the patella are also called epiphysoid bones and are developmentally equivalent to the epiphyses of the long bones.

Metaphysis: The metaphysis is the junctional region between the growth plate and the diaphysis. The metaphysis contains abundant trabecular bone, but the cortical bone thins here relative to the diaphysis. This region is a common site for many primary bone tumors and similar lesions.

Diaphysis: The diaphysis is the shaft of long bones and is located in the region between metaphyses, composed mainly of compact cortical bone. The medullary canal contains marrow and a small amount of trabecular bone.

Physis (epiphyseal plate, growth plate): The physis is the region that separates the epiphysis from the metaphysis. It is the zone of endochondral ossification in an actively growing bone or the epiphyseal scar in a fully grown bone.

TYPES OF BONE TISSUE

Bone tissue can be classified in several ways, based on texture, matrix arrangement, maturity, and developmental origin.

Based on texture of cross sections, bone tissue can be classified as follows:

Compact bone (dense bone, cortical bone): Compact bone is ivory like and dense in texture without cavities. It is the shell of many bones and surrounds the trabecular bone present in the center. Compact bone consists mainly of haversian systems or secondary osteons.

Spongy bone (trabecular bone, cancellous bone): Spongy bone is so named because it is sponge like with numerous cavities. It is located within the medullary cavity and consists of extensively connected bony trabeculae that are oriented along the lines of stress. In contrast to compact bone, complete osteons are usually absent in spongy bone due to the thinness of the trabeculae. Spongy bone is also more metabolically active than compact bone because of its much larger surface area for remodeling.

Based on matrix arrangement, bone tissue can be classified as follows:

Lamellar bone (secondary bone tissue): Lamellar bone is mature bone with collagen fibers that are arranged in lamellae. In contrast to spongy bone, in which lamellae are arranged parallel to each other, in compact bone, the lamellae are concentrically organized around a vascular canal, termed as haversian canal.

Woven bone (primary bone tissue): Woven bone is immature bone, in which collagen fibers are arranged in irregular random arrays and contain smaller amounts of mineral substance and a higher proportion of osteocytes than lamellar bone. Woven bone is temporary and is eventually converted to lamellar bone; this type of bone is also pathologic tissue in adults, except in a few places, such as areas near the sutures of the flat bones of the skull, tooth sockets.

Based on maturity, bone tissue can be classified as follows:

Immature bone (primary bone tissue): Immature bone is woven bone.

Mature bone (secondary bone tissue): Mature bone is characteristically lamellar bone. Almost all bones in adults are lamellar bones.

Based on developmental origin, bones can be classified as follows:

Intramembranous bone (mesenchymal bone): Intramembranous bone develops from direct transformation of condensed mesenchyme. Flat bones are formed in this way.

Intracartilaginous bone (cartilage bone, endochondral bone): Intracartilaginous bone forms by replacing a preformed cartilage model. Long bones are formed in this way.

The different parts of bone and their explanations are as follows (Fig. 1.1):

Periosteum: Periosteum is the highly vascular membranous tissue covering the bone that brings blood and lymph vessels, as well as nerves, to it. The functions of periosteum include: bone nutrition, longitudinal and transverse growth of bone, and its regeneration.

Periosteum has two layers:

- *External*—fibrous; made of dense irregular connective tissue
- *Internal*—cellular (osteogenic); contains many osteoblasts and blood vessels, some osteocytes as well.

Endosteum: It is a lining covering a bone from the marrow side, made of loose irregular connective tissue

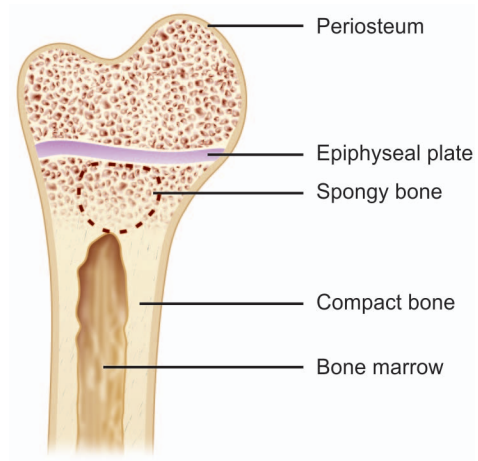


Fig. 1.1: Schematic representation of structure of a typical long bone

with osteoblasts and osteoclasts in addition to more common cell types of this tissue. It is a highly vascular condensation of areolar tissue lining the various medullary spaces

Compact bone (also known as cortical bone): Consists of dense deposits of minerals—chiefly calcium phosphate and Type I collagen. These are arranged in concentric circles around a central Haversian canal through which blood, lymph vessels as well as nerves pass through.

Spongy bone (also known as trabecular or cancellous bone): The mineral deposits are arranged as a system of struts. Bone marrow fills the spaces between.

Bone marrow: Some bones, such as the femur, also contain a central cavity filled with bone marrow. Bone marrow contains the stem cells that give rise to all the types of blood cells.

Epiphyseal plate: Until the end of puberty, this disk of cartilage produces more cartilage which then is converted into bone. In this way, the bone grows lengthwise.

MOLECULAR STRUCTURE OF BONE

Bone is created from osseous connective tissue. Like other types of connective tissue, osseous tissue is composed of relatively sparse cells surrounded by an extracellular network, or matrix. Bone matrix is a tough, resilient mixture of protein and minerals. Osteoblasts, a type of bone cell, secretes proteins into the matrix, which provide tensile strength (resistance to stretching and twisting). The principal protein of the bone matrix is collagen, which accounts for almost one-third of the dry weight of bone. Most of the rest of the bone's weight is due to the minerals of the matrix. These are mainly calcium phosphate and calcium carbonate. Embedded in the protein network, the minerals provide hardness and compressive strength.

Bone cells remain alive and, like other cells in the body, must be nourished by blood. In order to deliver nutrients and to remove waste from the bone interior, the hard, compact surface is pierced by "canals" through which blood vessels can travel. Once inside, these canals branch, allowing blood vessels to reach cells throughout the bone. This canal system gives bone its characteristic appearance under the microscope, with bone cells embedded in concentric rings (lamellae) of calcified

matrix, all surrounding a hollow canal. These units of structure, called osteons, run parallel in compact bone, but form a looser and less-ordered network in spongy bone. Compact bone forms in the perimeter of long bone shafts, such as those of the legs and arms, where stress forces tend to be in the same direction. In contrast, spongy bone is found in the ends of bones, where forces come from many different directions. Spongy bone also occurs where bone is not subject to significant stress.

The Matrix of Bone (Fig. 1.2)

Two types of bone matrix are observed in the mature skeleton: hard compact cortical bone, found largely in the shafts of the long bones that surround the marrow cavities; and spongy or cancellous bone, which comprises a network of fine, interlacing partitions, the trabeculae, enclosing cavities that contain either hematopoietic or fatty marrow. Cancellous bone is found in the vertebrae, in the majority of the flat bones, and in the ends of the long bones.

Cortical Bone

Cortical bone represents nearly 80 percent of the skeletal mass. It is also called compact bone, because it forms a protective outer shell around every bone in the body. Cortical bone has a slow turnover rate and a high resistance to bending and torsion. It provides strength

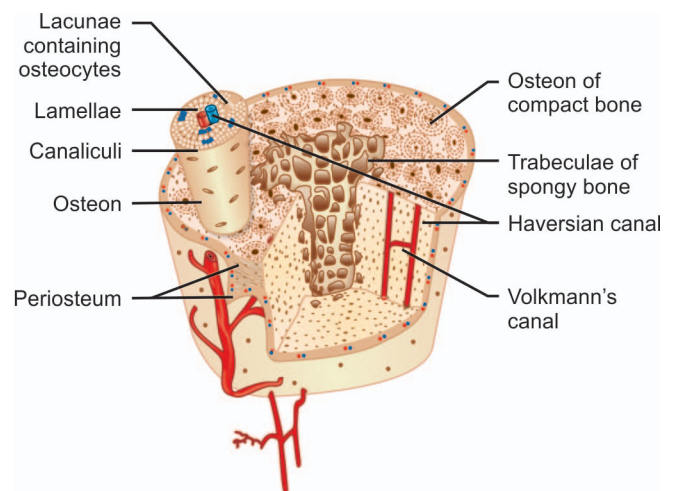


Fig. 1.2: Organization of bone: depiction of the lamellar bone in the shaft of a long bone, the Haversian systems, Volkmann's canal and inner and outer lamella

where bending would be undesirable as in the middle of long bones. A closer view at this kind of bone (Fig. 1.2) will show a series of adjacent and overlapping bull's eye formations called osteons or Haversian systems. Each osteon is composed of a central vascular channel surrounded by a kind of tunnel, called the Haversian canal. The canal can contain capillaries, arterioles, venules, nerves and possibly lymphatics. Between each osteon are interstitial lamellae (concentric layers of mineralized bone). Lamellar bone gets its strength from its plywood-like construction: parallel layers of bone alternate in orientation by 90 degrees. The Haversian canals communicate with the medullary cavity, with spaces in spongy bone and with the surfaces of bone by oblique or transverse channels termed Volkmann's canals.

Trabecular or Cancellous Bone

This kind of bone represents only 20 percent of the skeletal mass, but constitutes 80 percent of the bone surface. Trabecular bone is less dense, more elastic and has a higher turnover rate than cortical bone. It is found in the epiphyseal and metaphyseal regions of long bones and throughout the interior of short bones. Trabecular bone constitutes most of the bone tissue of the axial skeleton: bones of the skull, ribs and spine. It is formed in an intricate and structural mesh. Trabecular bone forms the interior scaffolding, which helps bone to maintain their shape despite compressive forces. Trabecular bone is rigid but appears spongy, it is composed of bundles of short and parallel strands of bone fused together. The trabeculae are arranged in a haphazard manner, but they are organized to provide maximum strength similar to braces that are used to support a building. The trabeculae of spongy bone follow the lines of stress and can realign if the direction of stress changes. The center of the bone contains red and yellow marrow, bone cells and other tissues.

Composition of Bone

The bony tissue consists of ground substance or matrix in which are embedded fibers and is impregnated with bone salts. The intercellular material, in which the cells and fibers of connective tissue are embedded, is composed largely of glycosaminoglycans, metabolites, water, and ions are called as ground substance. Water

constitutes about one-fifth of the weight of the matrix in mature bone; organic material forms 30-40 percent and mineral salts 60-70 percent of the dry weight.

The main organic components are collagen, mucopolysaccharides in combination with non-collagenous proteins. The uncalcified organic matrix is called osteoid matrix. The organic part of matrix is mainly composed of Type I collagen. This is synthesised intracellularly as tropocollagen and then exported. It then associates into fibrils. Also making up the organic part of matrix include various growth factors, the functions of which are not fully known. Other factors present include glycosaminoglycans, osteocalcin, osteonectin, bone sialoprotein and Cell Attachment Factor. One of the main things that distinguishes the matrix of a bone from that of another cell is that the matrix in bone is hard.

Collagens

Collagen is the most abundant protein in mammals. About one quarter of all of the protein in our body is collagen. Collagen is the main protein of connective tissue. It has great tensile strength, and is the main component of ligaments and tendons. It is responsible for skin elasticity, and its degradation leads to wrinkles that accompany aging. Collagen also fills out the cornea where it is present in crystalline form. It is also used in cosmetic surgery, for example lip enhancement. The triple helical structure of collagen was first proposed by Ramachandran's group from Madras. There are nearly 28 types of collagen described in literature. Over 90 percent of the collagen in the body are Collagens I, II, III, and IV. The general functions of collagens are: *Collagen I*—main component of bone and dentine, *Collagen II*—main component of cartilage, *Collagen III*—main component of reticular fibers, *Collagen IV*—forms the basement membrane.

A collagenous fiber is a bundle of many macrofibrils. Each macrofibril in turn is a bundle of numerous microfibrils. The microfibril is composed of many tropocollagen helices. Each of these are assembled from three polypeptide chains twisted together.

Collagen has an unusual amino acid composition. It contains large amounts of glycine and proline, as well as two amino acids that are not inserted directly by ribosomes—hydroxyproline and hydroxylysine—the

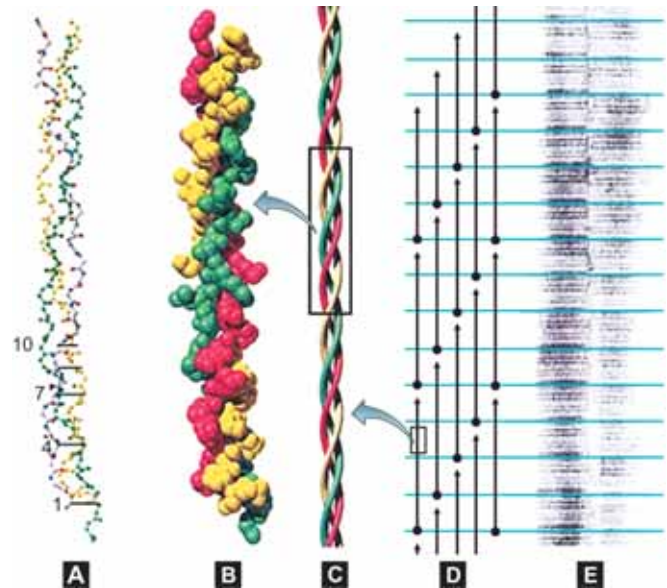
former composing a rather large percentage of the total amino acids. They are derived from proline and lysine in enzymatic processes of post translational modification, for which vitamin C is required. This is related to why vitamin C deficiencies can cause scurvy, a disease that leads to loss of teeth and easy bruising caused by a reduction in strength of connective tissue due to lack of collagen or defective collagen. The white collagen that makes up the matrix of most connective tissue in mammals consists of interwoven fibres of the protein collagen. The collagen fibres consist of globular units of the collagen sub-unit, the tropocollagen. Tropocollagen sub-units spontaneously arrange themselves under physiological conditions into staggered array structures stabilized by numerous hydrogen and covalent bonds. Tropocollagen sub-units are left-handed triple helices where each strand is, further, a right-handed helix by itself. Thus, tropocollagen may be considered to be a coiled coil. Each chain is left handed helix and the wrapping is right-handed.

Another rare feature of collagen is its regular arrangement of amino acids in each of the alpha chains of the collagen sub-units (Figs 1.3A to E). The sequence generally follows the pattern Gly-X-Y, where Gly for glycine, and X and Y for any amino acid residues. Most of the times, X is for proline and Y is for hydroxyproline. There are very few other proteins with such regularity. The inordinate number of Gly residues allows the otherwise sterically disallowed, tight coiling of each of the alpha chain subunits of tropocollagen, where there is a rise per turn of just 0.3 nm as opposed to the .36 nm of a regular Alpha helical coil. Hydroxylysine and hydroxyproline play important roles in the stabilization of the tropocollagen globular structure as well as the final fiber shaped structure by forming covalent bonds. The resulting structure is called a collagen helix.

The synthesis of type I collagen takes place in the following way (Figs 1.4A and B):

Inside the Cell

- Three peptide chains are formed (Two alpha-1 and one alpha-2 chain) in ribosomes along the Rough Endoplasmic Reticulum (RER). These peptide chains (known as procollagen) have registration peptides on each end; and a signal peptide is also attached to each.

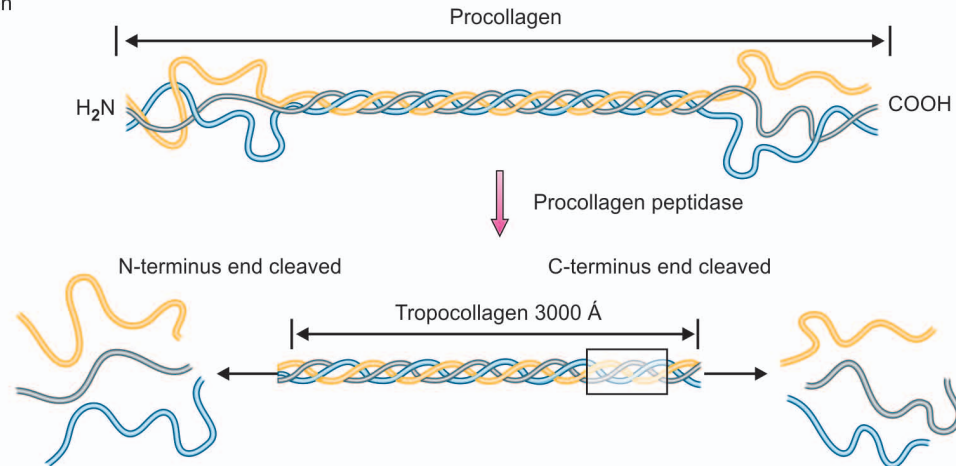
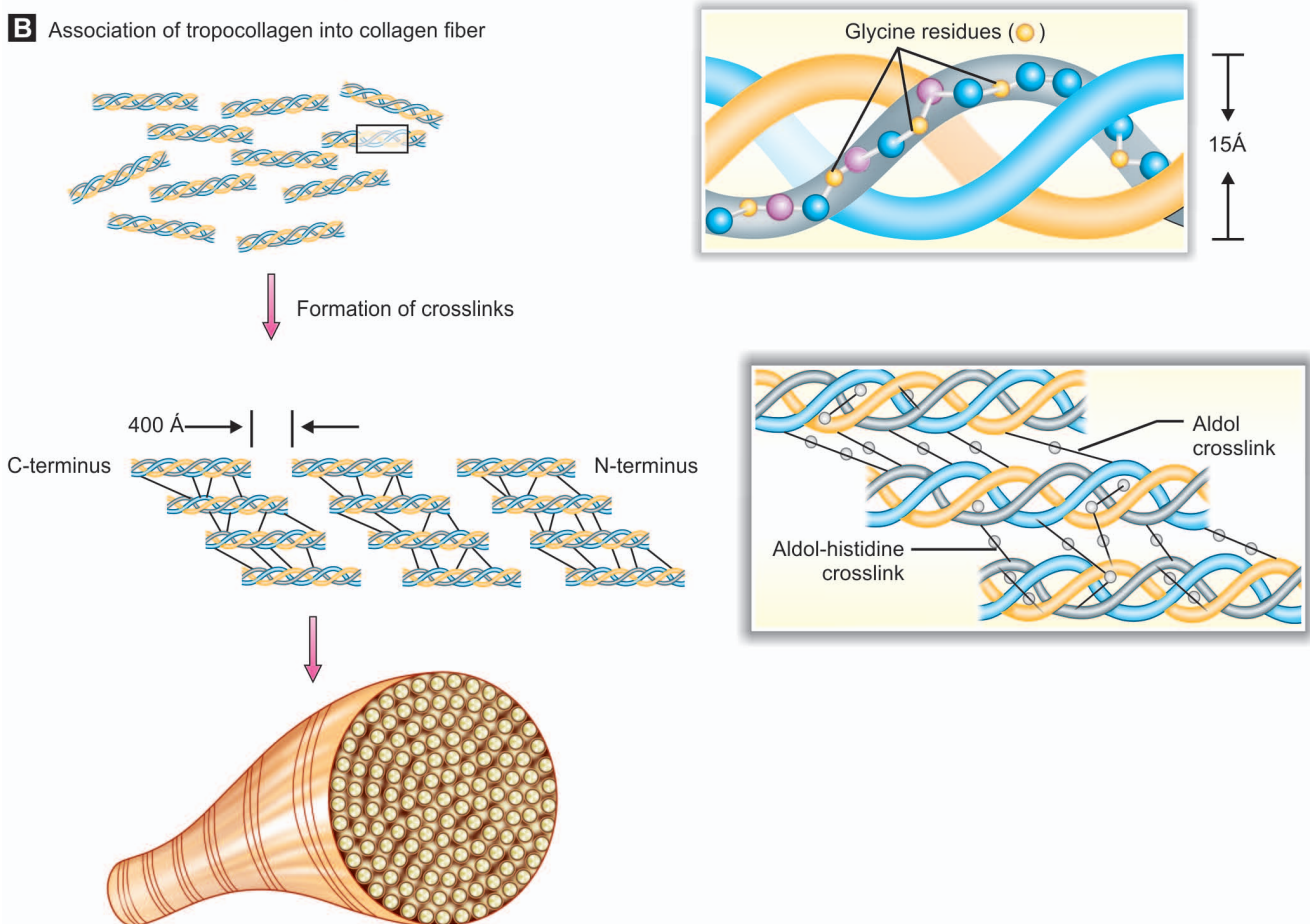


Figs 1.3A to E: The structure of collagen on several length scales. (A to C) The three subunits of collagen coil together into a triple helix, with the H side group of glycine fitting into the center of the molecule. Each subunit contains 1050 amino acids, and when wound the helix is about 300 nm long. (D and E) In a collagen fibril, adjacent collagen molecules are staggered with their ends 67 nm apart, producing visible striations in stained collagen

- Peptide chains are sent into the lumen of the RER.
- Signal Peptides are cleaved inside the RER and the chains are now known as procollagen.
- Hydroxylation of lysine and proline amino acids occurs inside the lumen. This process is dependent on Ascorbic Acid (Vitamin C) as a cofactor.
- Glycosylation of specific hydroxylated amino acid occurs.
- Triple helical structure is formed inside the RER.
- Procollagen is shipped to the golgi apparatus, where it is packaged and secreted by exocytosis.

Outside the Cell

- Registration peptides are cleaved and tropocollagen is formed by procollagen peptidase.
 - Multiple tropocollagen molecules form collagen fibrils, and multiple collagen fibrils form into collagen fibers.
 - Collagen is attached to cell membranes via several types of protein, including fibronectin and integrin.
- The collagen fiber is clearly identified in the electron microscope by the banded pattern which gets repeated

A Formation of tropocollagen**B** Association of tropocollagen into collagen fiber

Figs 1.4A and B: Synthesis of collagen (*Klug and Cummings, 1997*)

regularly at 64 nm intervals. The protein carbohydrate complexes of bone are of two types, proteoglycans and glycoproteins. These substances contribute to the physical characteristics of bone and cartilage. Proteoglycans consists of a protein chain or core to which is attached

polysaccharide side chains, glycosaminoglycans. The glycosaminoglycans consists of regularly repeating units of two sugars.

The inorganic part is mainly crystalline mineral salts and calcium, phosphate, hydroxyl ions, carbonate and

water which is present in the form of hydroxyapatite. The mineral is a basic calcium phosphate, being an apatite, hydroxylapatite $[\text{Ca}_{10}(\text{PO}_4)_6(\text{OH})_2]$.

Cells of Bone (Fig. 1.5)

Osteoblasts, osteocytes, osteoclasts, and osteogenic or osteoprogenitor cells are found in developing bone, regardless of the site of formation.

Osteoblasts

Osteoblasts take origin from poorly differentiated mesenchymal cells; residing in the internal layer of periosteum, during bone development, osteoblasts are on the periosteal surface and around interosseous blood vessels; these cells are cuboidal, columnar and polygonal in shape, have a well-developed rough endoplasmic reticulum. Osteoblasts are the cells responsible for the formation and organization of the extracellular matrix of bone and its subsequent mineralization. They are derived from mesenchymal precursor cells in marrow that have the potential to differentiate into fat cells, chondrocytes or muscle cells (Owen & Ashton, 1986; Beresford, 1989). The origin of osteoblastic cells in the developing long bones is less well defined. One hypothesis is that the osteoblasts are derived from blood-borne elements. This view is supported by evidence that cells in empty lacunae express type I collagen mRNA and are morphologically similar to osteoblasts, but unlike hypertrophic chondrocytes do not express type X collagen mRNA. The alternative view is that osteoblasts are derived from hypertrophic chondrocytes, since type I collagen has been immunolocalized in apparently intact lacunae (don der Mark, 1989). Osteoblasts form a cell

layer over bone surfaces on which matrix is being formed. The cells are polarized, in that new osteoid, referred to as an osteoid seam, is deposited along the surface adjacent to bone. The deeper portion of the osteoid seam undergoes mineralization along the so-called mineralization front. The bone is essentially enveloped by the osteoblasts, since the cells are in close contact with one another and tight junctions and gap junctions have been observed. Thus, the osteoblastic layer controls the transport of materials from the extracellular space to the osteoid seam and mineralization front. Ultrastructurally, osteoblasts (Fig. 1.6) feature a complement of organelles characteristic of cells actively involved in protein synthesis. They have abundant endoplasmic reticulum and numerous ribosomes, and the Golgi apparatus and mitochondria are quite prominent. Procollagen molecules are produced by the ribosomes and extruded into the extracellular space, but only along the surface that faces bone. Proteolysis and polymerization within the extracellular space results in the formation of collagen fibrils. The combination of these intracellular and extracellular events leads to the production of the osteoid seam. Most of the proteoglycans are packaged in the Golgi apparatus, and vesicles containing these products then migrate to the surface of the cell and release their contents by exocytosis.

Membrane-bound vesicles containing amorphous calcium phosphate are extruded from the plasma membrane of the osteoblast into the extracellular space. It appears that these vesicles induce and actually activate crystal formation. Alkaline phosphatase, which is produced by the osteoblast, is thought to act as a pyrophosphatase and may be involved in the initiation

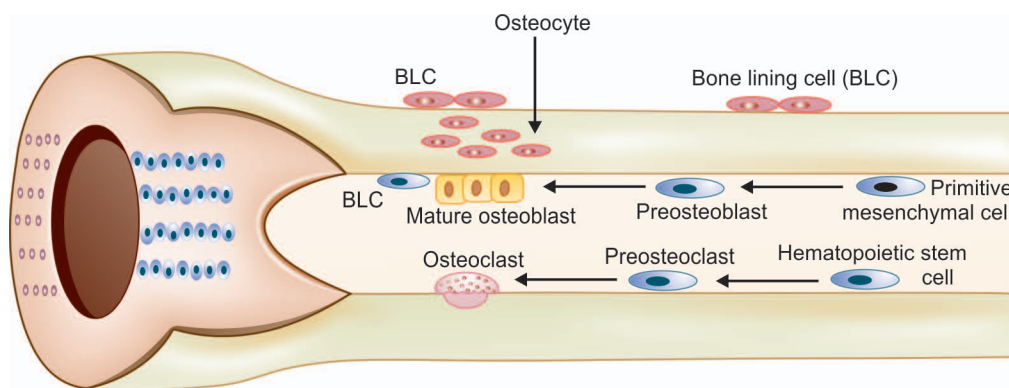


Fig. 1.5: Diagrammatic representation of cells present in bone

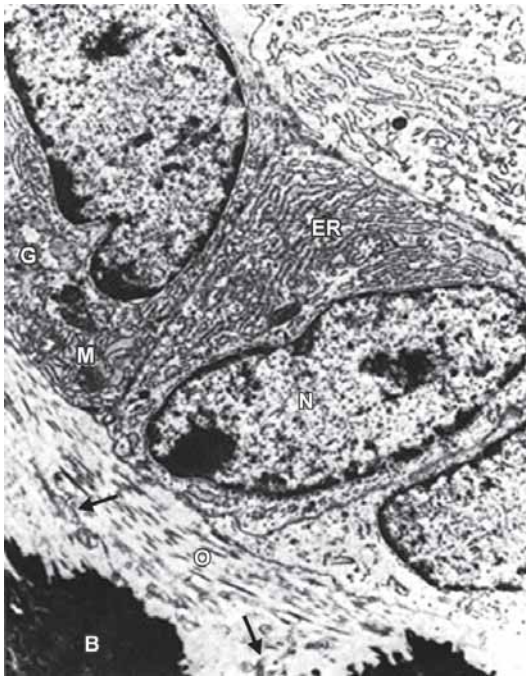


Fig. 1.6: Osteoblasts lining a trabeculum of cancellous bone. The cytoplasm contains rough endoplasmic reticulum (ER) and scattered mitochondria (M). Golgi complexes (G) are well developed and have cisternae distended with fibrillar material. Cytoplasmic processes of osteoblasts (arrows) extend into adjacent osteoid (O), which is interposed between the osteoblasts and underlying mineralized matrix of woven bone (B). Osteoblast nuclei are shown (N). (original magnification $\times 6420$) [Fetter AW. *Electron microscopic evaluation of bone cells in pigs with experimentally induced Bordetella rhinitis (turbinate osteoporosis)*. Am J Vet Res 1975;36(1):15-22]

of the mineralization process. The transformation of amorphous calcium phosphate to crystalline hydroxyapatite appears to take place both inside and outside the matrix vesicles. When crystals formed within the vesicle contact the membrane of the vesicle, the membrane ruptures. The crystals, which are then exposed to a supersaturated solution, induce precipitation over the entire matrix surface.

The principal products of the mature osteoblast are type I collagen (90% of the protein in bone), the bone specific vitamin-K dependent proteins, osteocalcin and matrix Gla protein, the phosphorylated glycoproteins including bone sialoproteins I and II, osteopontin and osteonectin, proteoglycans and alkaline phosphatase.

Osteocytes

A mature form of osteoblasts, they lie in lacunae within a bone and extend protoplasmic processes into small canaliculi in the intercellular matrix. Approximately 10 percent of the osteoblastic population become enclosed in the developing matrix and are then referred to as osteocytes. Osteocytes have structural features very similar to osteoblasts when they are on the surface of the matrix, but the endoplasmic reticulum may not be so profuse. As the cells become more deeply embedded in mineralized bone matrix, their cytoplasmic volume is reduced, as is their complement of cytoplasmic organelles. Osteocytes have cytoplasmic processes that extend into the surrounding matrix for some distance and fill most of the canaliculi in which they are contained. The processes of osteocytes contact processes from other osteocytes and osteoblasts on the surface, forming tight junctions. This interconnection of osteoblastic lining cells with the osteocytes deep within bone regulates the flow of mineral ions from the extracellular fluid through the osteoblast to the osteocytes, from the osteocytes to the extracellular fluid surrounding them, and finally from this fluid to the mineralized bone matrix. Thus, the large surface area provided by the osteocytic population results in a regulatory mechanism for the exchange of mineral ions between the extracellular fluid and bone by means of the canalicular system. Osteocytes appear to be essential to the maintenance of bone, since when the cell dies, the matrix around it eventually is removed. Osteocytes have also been shown to act as mechano-sensory receptors—regulating the bone's response to stress and mechanical load. They are mature bone cells.

Osteoclasts

Osteoclasts are large, multinucleated cells located on bone surfaces in what are called Howship's lacunae or resorption pits. These lacunae, or resorption pits, are left behind after the breakdown of bone and often present as scalloped surfaces. Osteoclasts are macrophages of bone tissue, blood monocytes being their precursors; large multinucleated cells; a zone of cytoplasm adjacent to osseous surface is referred to as ruffled border, multiple cytoplasmic processes and lysosomes are found in them. The size of osteoclasts may be up to $200,000 \mu\text{m}^3$ with up to 100 nuclei. Functions of osteoclasts include destruction and resorption of bone

fibers and ground substance. Because the osteoclasts are derived from a monocyte stem-cell lineage, they are equipped with engulfment strategies similar to circulating macrophages. Osteoclasts mature and/or migrate to discrete bone surfaces. Upon arrival, active enzymes, such as tartrate resistant acid phosphatase, are secreted against the mineral substrate.

Osteoclasts are polarized cells, having a ruffled border region of the cell membrane that is surrounded by an organelle-free region, or 'clear zone', and they adhere to the bone surface via integrins, which are specialized cell surface receptors. Osteoclastic bone resorption initially involves mineral dissolution, followed by degradation of the organic phase. These processes take place beneath the ruffled border and depend on lysosomal enzyme secretion and an acid microenvironment. A pH gradient across the ruffled membrane is the consequence of active transport mechanisms such as Na^+/H^+ exchange, ATP-dependent proton pumps, and the enzyme carbonic anhydrase. Osteoclasts actively synthesize lysosomal enzymes, in particular the tartrate resistant isoenzyme of acid phosphatase (TRAP) (used as a marker of the osteoclast phenotype), and cysteine-proteinases such as the cathepsin S that are capable of degrading collagen. Lysosomal enzymes are only released at the ruffled border region of the osteoclast cell membrane. Other cells in bone, in particular the osteoblast, may be involved in degrading the organic non-mineralized phase from bone surfaces. *In vitro* studies have shown that removal of non-mineralized organic matrix is necessary before mineralized matrix may be resorbed by isolated osteoclasts (Chambers & Fuller, 1985).

Systemic agents, important in regulating osteoclastic bone resorption, are parathyroid hormone (PTH), 1,25 di-hydroxy vitamin D_3 [$1,25(\text{OH})_2\text{D}_3$] and calcitonin. PTH and $1,25(\text{OH})_2\text{D}_3$ are unable to stimulate osteoclastic bone resorption *in vitro* in the absence of osteoblastic cells. This gave rise to the idea that these agents stimulate osteoclasts to resorb bone via a 'coupling' factor. Osteoclasts do not have receptors for $1,25(\text{OH})_2\text{D}_3$, and until recently were not believed to have PTH receptors, although the functional significance of PTH receptors on osteoclasts remains to be established. Osteoclasts have calcitonin receptors and this inhibitor of bone resorption acts directly on the osteoclast to reduce cellular motility, retract cytoplasmic extensions

and reduce ruffled border size. Glucocorticoids (GCs) are another class of systemic agents that cause bone loss. Although this may be due to their inhibition of intestinal calcium absorption and the induction of secondary hyperparathyroidism, GCs also have direct actions on bone cells. These direct effects on bone are believed to be via the local regulation of cytokine and prostaglandin production. Prostaglandins are locally produced by most cells in the body, and have been shown to have direct effects on osteoclasts and their precursors, inhibiting bone resorption by mature osteoclasts and increasing the formation of their precursors.

There is no clear evidence as to the fate of osteocytes when bone matrix is resorbed. It has been suggested that osteoclasts can undergo fission into mononuclear cells on the endosteal bone surface and that these mononuclear cells undergo a modulation of cell function to osteoblasts and then osteocytes.

Osteogenic Cells

Osteogenic cells are derived from undifferentiated mesenchymal cells, which are also believed to give rise to the formed hematopoietic elements. There is evidence that bone marrow contains both predetermined osteoprogenitor cells and cells that will form bone in the presence of a suitable inducer.

SKELETOGENESIS/BONE FORMATION

Bone formation occurs by three co-ordinated processes: the *production* and the *maturation* of osteoid matrix or skeletogenesis, and subsequent *mineralization* of the matrix. In the embryo, bone tissue arises through two processes, intramembranous ossification and endochondral ossification. In intramembranous ossification, bone is formed directly from mesenchymal tissue. The flat bones of the skull and face, the mandible and the clavicle develop in this manner. In endochondral ossification, a cartilage model of the bone is formed first, and is later replaced by bone. The weight-bearing bones of the axial skeleton and the bones of the extremities (most of the skeleton) develop in this manner.

The first bone to arise, whether from mesenchyme or from cartilage (or in fracture repair postnatally), is in the form of spicules. These first spicules are made of immature bone, also called woven bone. In immature bone, the collagenous lamellae are not arranged in

parallel or concentric arrays (as in mature spongy and compact bone, respectively), but are randomly oriented and loosely intertwined (hence woven). Immature bone also has more ground substance than mature bone. Consequently, immature and mature bone (Fig. 1.7) show different staining characteristics, immature bone stains more with hematoxylin and mature bone more with eosin. The spicules of immature bone are remodeled. The remodeling process can eventually give rise to more spongy bone or to compact bone. The remodeling of bone continues throughout life (remember those interstitial lamellae in compact bone represent former osteons). Immature bone is the predominant bone in the developing fetus. In the adult, most immature bone is replaced by mature bone, but immature bone is seen where bone is being remodelled or repaired, and in certain specific areas, such as the alveolar sockets of the oral cavity. When bone matrix is first secreted, it is not yet mineralized and is called osteoid. As mentioned above, osteoblasts also bring about the mineralization of bone.

Membranous Ossification (Fig. 1.8)

Also called as direct ossification/intramembranous ossification. The first step in intramembranous ossification is the aggregation of mesenchymal cells in the area where

bone is to be formed. The tissue in this area becomes more vascularized, and the mesenchyme cells begin to differentiate into osteoblasts, which secrete the collagen and ground substance (proteoglycans) of bone matrix (collectively called osteoid). The osteoblasts maintain contact with one another via cell process. The osteoid becomes calcified with time, and the processes of the cells (called osteocytes when they are surrounded with matrix) become enclosed in canaliculi. Some of the mesenchymal cells surrounding the developing bone spicules proliferate and differentiate into osteoprogenitor cells. Osteoprogenitor cells in contact with the bone spicule become osteoblasts, and secrete matrix, resulting in appositional growth of the spicule. Intramembranous ossification begins at about the eighth week in the human embryo.

The sequence of events which take place in membranous ossification are as follows:

- Increased vascularity of tissue.
- Active proliferation of mesenchymal cells. The mesenchymal cells give rise to osteogenic cells, which develop into osteoblasts.
- Osteoblasts begin to lay down osteoid. Osteoid is the organic part of bone without the inorganic constituent.
- Osteoblasts either retreat or become entrapped as osteocytes in the osteoid.

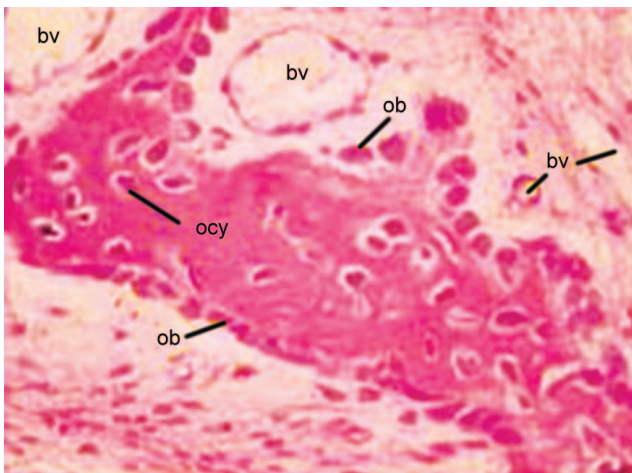


Fig. 1.7: Numerous osteocytes (ocy) are seen enclosed within the spicule, and most of the periphery is lined by osteoblasts (ob). (A characteristic feature of immature bone is a greater abundance of cells than in mature bone). Several developing blood vessels (bv) are seen in the mesenchyme

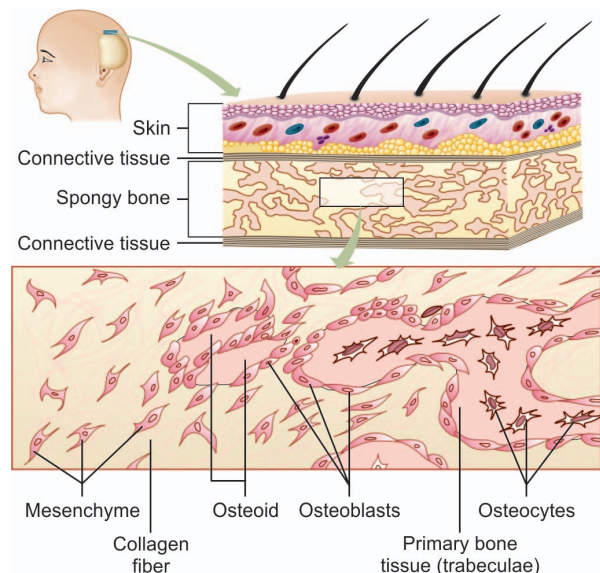


Fig. 1.8: Intramembranous Ossification (Source: Gartner and Hiatt, Color Textbook of Histology)

- The osteoid calcifies to form spicules of spongy bone. The spicules unite to form trabeculae. The inorganic salts carried in by the blood vessels supposedly bring about calcification. The salts are deposited in an orderly fashion as fine crystals (hydroxyapatite crystals) intimately associated with the collagenous fibers. These crystals are only visible with the electron microscope.
- Bone remodeling occurs. Periosteum and compact bone are formed.

Intramembraneous bone formation occurs on the outer surface of periosteum, the Endosteum, on the surfaces of trabeculae of cancellous bone and at the edges in specialized structures called sutures.

Endochondral Ossification

Endochondral ossification is the replacement of hyaline cartilage by bone. It is usually evident in the long bone, but the growth of cranial base synchondroses and condylar cartilage also serves the purpose equally. Endochondral ossification also begins with the aggregation of mesenchyme cells, but these differentiate into chondroblasts which secrete hyaline cartilage matrix. The cartilage is secreted in the general shape of the bone that it will become, and grows by both interstitial (mostly in length) and appositional (mostly in width) growth.

Sometimes, during the growth of this cartilage model (starting at about week 12 in the human fetus), some of the inner perichondrial cells begin to give rise to osteoblasts instead of chondroblasts (As a result, the former perichondrium is now called the periosteum). In long bones, this process begins at the mid-region of the bone. The newly formed osteoblasts secrete osteoid, forming a bone collar around the cartilage model. Therefore the very first bone that is formed during endochondral ossification is considered to arise by intramembraneous ossification.

Development of long bones begins with condensation of the mesenchyme to form a cartilaginous model of the bone to be formed (Fig. 1.9). Mesenchymal cells undergo division and differentiate into prechondroblasts and then into chondroblasts. These cells secrete the cartilaginous matrix. Like osteoblasts, the chondroblasts become progressively embedded within their own matrix, where they lie within lacunae, and they are then called chondrocytes. Unlike osteocytes however, chondrocytes continue to proliferate for some time, this being allowed in part by the gel-like consistency of cartilage. At the periphery of this cartilage (the perichondrium), the mesenchymal cells continue to proliferate and differentiate. This is called appositional growth. Another type of growth is observed in the

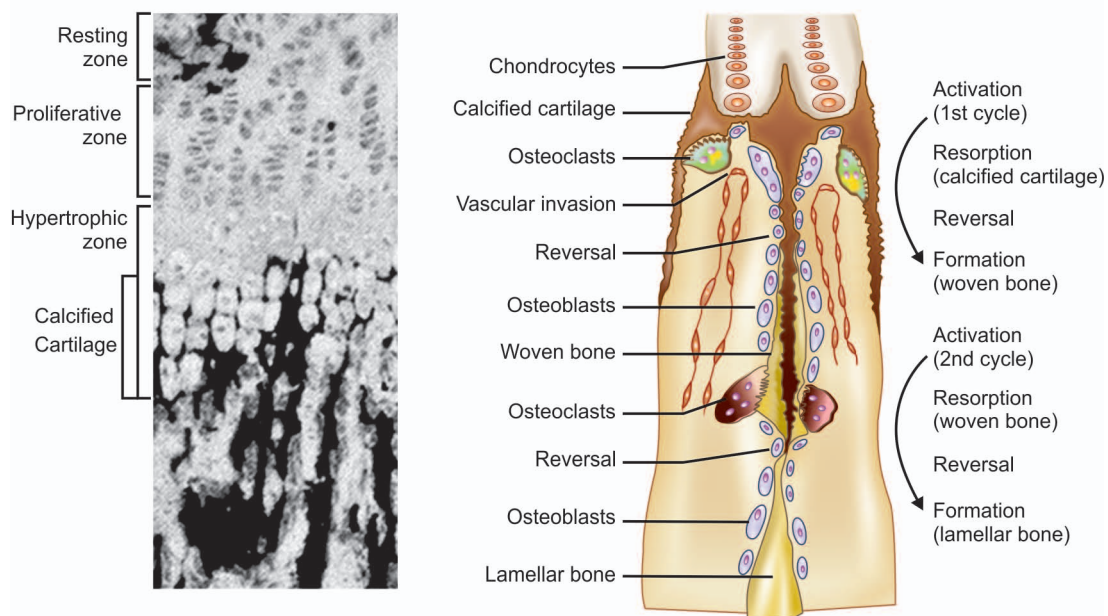


Fig. 1.9: Postnatal development of long bones. The four layers namely resting or zone of reserve cells, zone of hyperplasia or proliferation, zone of hypertrophy and zone of matrix calcification are seen

cartilage by cell proliferation and synthesis of new matrix between the chondrocytes (interstitial growth).

Beginning in the center of the cartilage model, at what is to become the primary ossification center, chondrocytes differentiate and become hypertrophic. During this process, hypertrophic cells deposit a mineralized matrix, where cartilage calcification is initiated by matrix vesicles. Once this matrix is calcified, it is partially resorbed by osteoclasts. After resorption and a reversal phase, osteoblasts differentiate in this area and form a layer of woven bone on top of the remaining cartilage. This woven bone will later be remodeled into lamellar bone.

The embryonic cartilage is avascular. During its early development, a ring of woven bone is formed at the periphery by intramembranous ossification in the future midshaft area under the perichondrium (which becomes periosteum). Following calcification of this woven bone, blood vessels, preceded by the osteoclasts entering the primary ossification center, will penetrate this bone and the calcified cartilage, forming the blood supply which will allow seeding of the hematopoietic bone marrow and invasion of osteoclasts to resorb the calcified cartilage.

Secondary ossification centers begin to form at the epiphyseal ends (Fig. 1.10) of the cartilaginous model, and by a similar process, trabecular bone and a marrow space are formed at these ends. Between the primary and secondary ossification centers, epiphyseal cartilage (growth plates) remain until adulthood. The continued differentiation of chondrocytes, cartilage mineralization and subsequent remodeling cycles allow longitudinal bone growth to occur, such that as new bone is formed, the bone will reach its final adult shape. There is, however, a progressive decrease in chondrocyte proliferation so that the growth plate becomes progressively thinner, allowing mineralization and resorption to catch up. It is at this point that the growth plates are completely remodeled and longitudinal growth is arrested.

The growth plate demonstrates, from the epiphyseal area to the diaphyseal area, the different stages of chondrocyte differentiation involved in endochondral bone formation (see Fig. 1.9). Firstly, a proliferative zone, where the chondroblasts divide actively, forming isogenous groups, and actively synthesizing the matrix. These cells become progressively larger, enlarging their lacunae in the pre-hypertrophic and hypertrophic zones. Lower in this area, the matrix of the longitudinal cartilage

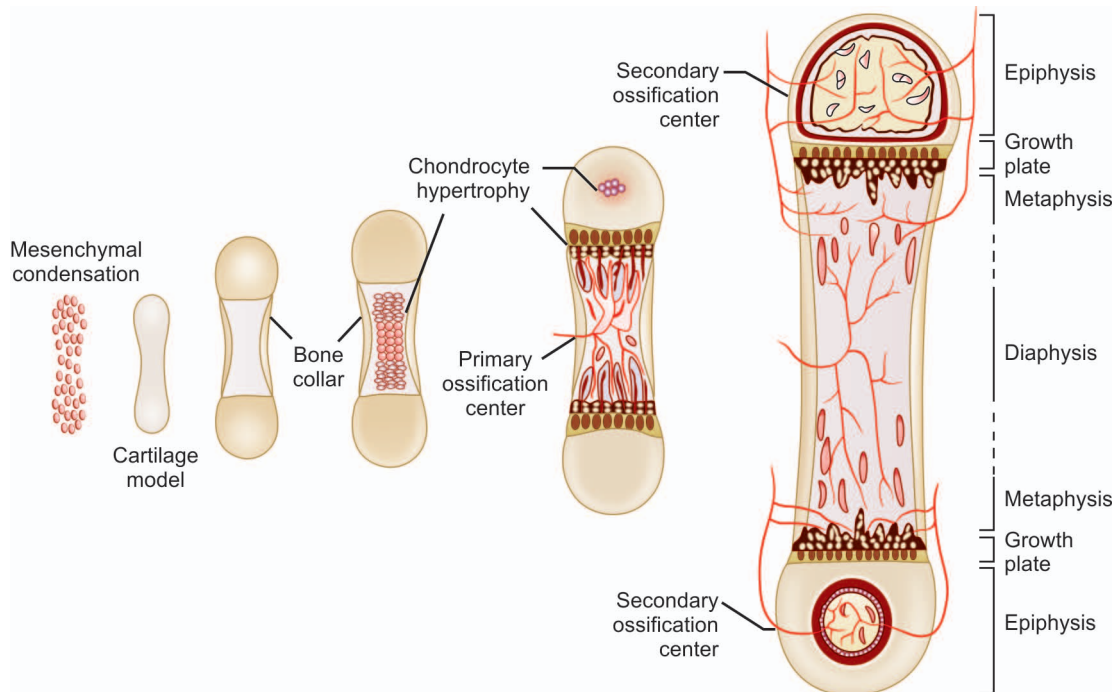


Fig. 1.10: Prenatal long bone development: Steps in the conversion of cartilage anlage to a mature long bone. Primary and secondary ossification centers are evident

septa selectively calcifies (zone of provisional calcification). The chondrocytes become highly vacuolated and then die through programmed cell death (apoptosis). Once calcified, the cartilage matrix is resorbed, but only partially, by osteoclasts, leaving the calcified longitudinal septae, and blood vessels appear in the zone of invasion. After resorption, osteoblasts differentiate and form a layer of woven bone on top of the cartilaginous remnants of the longitudinal septa. Thus, the first ARF sequence is complete: the cartilage has been remodeled and replaced by woven bone. The resulting trabeculae are called the primary spongiosum. Still lower in the growth plate, this woven bone is subjected to further remodeling (a second ARF sequence) in which the woven bone and the cartilaginous remnants are replaced with lamellar bone, resulting in the mature trabecular bone called secondary spongiosum.

Chondrocyte differentiation is regulated by a number of factors which have recently been described. The first factor shown to control chondrocyte differentiation was parathyroid hormone related peptide (PTHrP). This factor prolongs chondrocyte proliferation, and in PTHrP knockout mice, the main phenotype is bone shortening caused by premature chondrocyte hypertrophy. Targeted overexpression of PTHrP results in the opposite phenotype, with prolonged delay in chondrocyte maturation. PTHrP is part of a genetic signaling cascade, where not only is it regulated by factors expressed earlier in chondrocyte differentiation, such as Indian hedgehog (Ihh), but it also regulates chondrocyte differentiation itself, and alters gene expression in more mature chondrocytes. Other factors which regulate chondrocyte differentiation include the FGFs and bone morphogenetic proteins (BMPs). Table 1.1 shows the bones of skull and their modes of ossification.

Initiation of Calcification or Mineralization

The matrix is initially laid down as unmineralized osteoid (manufactured by osteoblasts). Mineralization involves osteoblasts secreting vesicles containing alkaline phosphatase. This cleaves the phosphate groups and acts as the foci for calcium and phosphate deposition. The vesicles then rupture and act as a centre for crystals to grow on. The exact mechanism of calcification is still under debate. Robinson noticed that alkaline phosphatase was always present at sites of calcification and suggested that this enzyme hydrolyzed phosphate esters to increase the

Table 1.1: Mode of ossification of the bones of the skull

<i>Membrane</i>	<i>Cartilage</i>
Maxilla	Ethmoidal bone
Zygomatic bone	Nasal concha
Palatine bone	Sphenoidal base
Vomer	Petrous part of temporal bone
Lacrimal bone	Occipital basalis
Frontal bone	Malleus
Parietal bone	Incus
Squamous and petrous part of temporal bone	Stapes
Squamous occipitalis	
Os Sphenoidal	
• Lamina medialis	
• Apex of ala major	

local concentration of phosphate to the level where calcium phosphate would precipitate. The second concept "seeding concept" suggests that a crystalline substance by virtue of its structure is similar to hydroxyapatite to induce the aggregation of calcium and phosphate ions. The body fluids which are supersaturated would then form a complete crystal by oriented growth or epitaxy. The seed may be organic or crystalline in nature. Electron microscopic studies have shown that the earliest resolvable mineral particles that can be seen are associated with the periodic banding of the collagen fibers. The third concept states that initiation of calcification occurs by the production of so called "matrix vesicles". The vesicles are produced by the cells of calcifying systems by a process of budding from the cell membrane. Vesicles are produced with the ability to form apatite crystals internally which eventually rupture the enclosing membrane and are exposed to the tissue fluids resulting in further crystallite growth. These vesicles are identified in all calcifying tissues except enamel.

Factors Influencing Mineralization

Local Factors

Collagen—Collagen provides an oriented support for newly formed mineral crystals. The specific longitudinal and lateral staggering of the collagen molecules in the fibril results in holes and pores in which nucleation, crystal growth, secondary nucleation and multiplication of the solid phase can occur.

Noncollagenous molecules

Name	Composition	Possible Function
Osteopontin	Phosphoprotein	Inhibits crystal growth
Osteonectin	Phosphoprotein	Inhibits crystal growth
Bone sialo-protein	Phosphorylated glycoprotein	Nucleator for mineralization
GLA Protein	Protein and γ -carboxy glutamic acid	Regulator of crystal growth
Biglycan and Decorin	Chondroitin sulfate and proteoglycans	Removed at mineralization front to permit mineralization
	Phospholipids	Calcium binding at mineralization front
	Pyrophosphate	Inhibitor of calcification

Growth Factors	
FGF	Increase osteoblastic precursor population and also increase collagen synthesis
IGF	Increase bone cell proliferation and total protein synthesis
TGF, PGDF	: Increase proliferation of osteoprogenitor and total protein synthesis
Interleukin 1	: At low doses, it stimulates collagen synthesis but is inhibitor in higher concentrations

Tumor necrosis factor: stimulate proliferation and collagen synthesis in preosteoblasts.

Systemic factors: Parathyroid hormone, 1, 25 - dihydroxy vitamin D₃, estrogen, insulin, etc. exerts effect on bone formation.

Role of alkaline phosphatase: Alkaline phosphatase is an ecto enzyme produced by osteoblasts that is a useful marker of osteoblast activity. It has a clear function—hydrolyzing phosphate ions from organic radical at an alkaline pH. Its precise role in mineralization is not clear. Hydroxyapatite crystals in contact with serum or tissue fluids are prevented from growing larger because pyrophosphate ions are deposited on their surfaces inhibiting further growth. Alkaline phosphatase activity breaks down pyrophosphate thereby permitting crystal growth to proceed.

MECHANISM OF BONE GROWTH

There are three basic mechanisms by which growth takes place at the cellular/tissue level. They are:

- **Hyperplasia:** Growth due to increase in number of cells.
- **Hypertrophy:** Growth due to increase in size of the cells.

- **Extracellular Matrix Secretion:** In this process, there is increase in size because of the secretions of the cells, into the extracellular matrix.

In hard tissues like bone and teeth, the extracellular material gets mineralized. Because of mineralization, interstitial growth is not possible in hard tissue. Hyperplasia, hypertrophy and extracellular matrix secretion all occur only on the surface. New cell formation takes place in the periosteum, the soft tissue membrane that covers the bone. Therefore, bone growth takes place only by "Surface deposition of bone". There is addition of fresh bone to the surface of existing bone.

Post natal growth of bone takes place in the following three ways:

- **Chondral growth,** achieved by interstitial growth of cartilage originating in cartilage. Example includes synchondroses.
- **Sutural growth** is appositional growth and occurs in the skull and facial sutures on the edge of bones.
- **Periosteal growth** which is also appositional and occurs in the periosteum. Periosteal growth unlike the chondral and sutural growth continues into advanced age.

Chondral Growth

Cartilage occurs at the base of skull in the form of synchondroses, nasal septum, symphyseal and condylar cartilage. Synchondroses growth continues until the bone is ossified. The significance of synchondroses are as follows:

- The organic matrix in cartilage is not normally calcified and because the tissue is avascular, metabolites must enter and leave by simple diffusion. As there are no blood vessels to occlude, cartilage can be utilized in areas of the skeleton which are subjected to pressure.
- Also cartilage like soft tissue has interstitial as well as appositional growth. Interstitial growth is possible because the matrix is not calcified and can therefore expand to accommodate the chondrocytes resulting from cell division.
- The physical and biological properties of cartilage are ideal for flexible support and growth necessary during skeletogenesis.
- The growth of cartilage is also bidirectional unlike the periosteal growth which is unidirectional.

The growth and activity of synchondroses is controlled by growth hormones (STH). Excessive production of

STH results in lengthening of cranial base, while under production leads to shortening of base of skull and hence leads to underdevelopment of midface. The importance of nasal septum is disputable. The cartilage at the symphysis menti retains its growth activity up to first year of life whereas condylar cartilage growth activity is traced up to maturity.

Sutural Growth

Sutural growth occurs due to osteoblasts and is similar to periosteal growth, the difference being that bone apposition takes place at the edges of bone. The histology of suture shows, a cellular osteoblastic layer bordering the bone, a fibrous layer and a middle zone (Fig. 1.11). The middle zone contains numerous blood vessels and connects both the fibrous layer to one another. The active growth of suture is found only at the bone edges. Premature fusion (synostosis) of suture leads to skull deformities. Table 1.2 shows the different deformities produced due to premature fusion of craniofacial sutures.

The various bony joints are given as follows:

Bony joints:

- **Suture:** Type of fibrous joint in which the opposed surfaces are firmly united.
- **Symphysis:** Two bony surfaces are firmly united by a plate of fibro cartilage.
- **Synostosis:** Union of adjacent bones by osseous matter.
- **Syndesmosis:** Fibrous junction in which the intervening fibers form an interosseous membrane.
- **Synchondrosis:** A cartilaginous joint that is usually temporary and gets converted into bone in adult life.

Scott and Dixon (1978) have summarized the craniofacial sutures system as follows:

- Lambdoid suture system divides the occipital squamous from the parietal and temporal bones. Growth from the suture affects mainly the back of skull.
- Coronal suture promote longitudinal growth of the skull.
- Craniofacial and maxillofacial suture system contribute to forcing the middle face downwards and forwards.
- Sagittal suture is responsible for growth in width of the cerebral and facial skull.

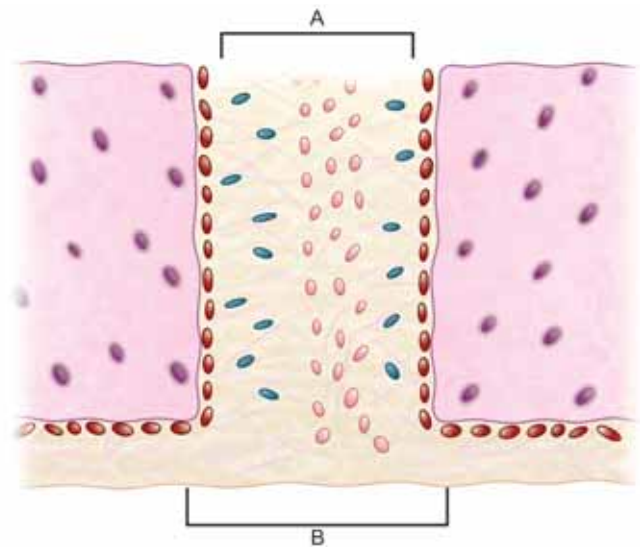


Fig. 1.11: A schematic illustration of the two differing views on the structure of the suture. A represents the three-layer concept; B, the five-layer concept

Table 1.2: Premature closure of various sutures and their effects

Sutures	Skull deformity
Sagittal suture	Scaphocephaly
Symmetric fusion of coronal suture	Oxycephaly (Tower skull)
Asymmetric fusion of coronal suture	Plagiocephaly
Metopic suture	Trigonocephaly (wedge skull)

Periosteal Growth

Periosteum controls the resorption and deposition of the bone during maturity. The growth direction of periosteal growth is on one side only and bone growth or deposition takes place only on the surface.

Remodeling

Bone remodeling is the process by which bone is turned over; it is the result of the activity of the bone cells at the surfaces of bone, mainly the endosteal surface (which includes all trabecular surfaces). Remodeling is traditionally classified into two distinct types: *Haversian remodeling* within the cortical bone and *endosteal remodeling* along the trabecular bone surface. This

distinction is more morphological than physiological because the Haversian surface is an extension of the endosteal surface and the cellular events during these two remodeling processes follow exactly the same sequence.

Bone formation and bone resorption do not occur along the bone surface at random: they are co-ordinated as part of the turnover mechanism by which old bone is replaced by new bone. In the normal adult skeleton, bone formation only occurs, for the most part, where bone resorption has already occurred. This basic principle of cellular activity at the remodeling site is the Activation-Resorption-Formation (ARF) sequence (Fig. 1.12).

Under some signal, a locally acting factor released by lining cells, osteocytes, marrow cells, or in response to bone deformation or fatigue-related microfracture, a group of preosteoclasts are activated. These mononuclear cells attach to the bone via $\alpha_v\beta_3$ integrins and fuse to form a multi-nucleated osteoclast which will, in a definite area of the bone surface, resorb the bone matrix. After resorption of the bone, and osteoclast detachment, uncharacterized mononuclear cells cover the surface and a cement line is formed. This is termed the reversal phase, and is followed by a period of bone formation. Preosteoblasts are activated, proliferate and differentiate into osteoblasts, which move onto the bone surface, forming an initial matrix (osteoid), which becomes mineralized after a time lag (the osteoid maturation period). The basic remodeling sequence is therefore Activation-Resorption-Formation; it is performed by a group of cells called the Basic Multicellular Unit (BMU). The complete remodeling cycle takes about 3 months in humans.

CARTILAGE

Cartilage is a solid connective tissue that is to a certain extent pliable, making it resilient. These characteristics of cartilage are due to the nature of its matrix. The ground substance of cartilage is rich in proteoglycans (Fig. 1.13) consisting of a core protein with numerous—about 100—glycosaminoglycans (GAGs) attached in bottle-brush fashion around it. GAGs are made of repeating units of disaccharides, one of which is always a glycosamine (hence the name) such as glucosamine or galactosamine (Glycosamines are also called

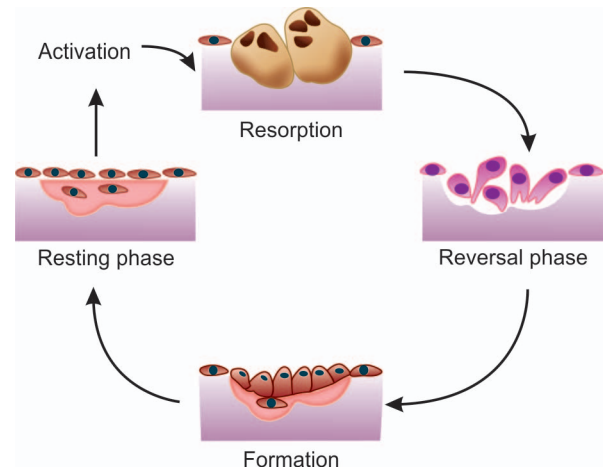


Fig. 1.12: The ARF sequence of bone remodeling

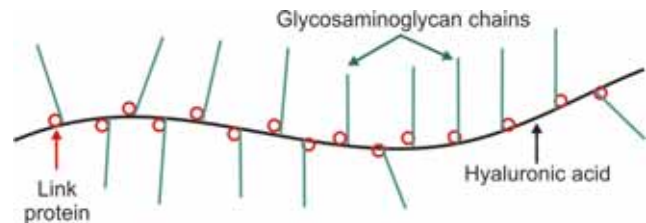


Fig. 1.13: Diagrammatic representation of structure of cartilage proteoglycans

hexosamines). In cartilage, the GAGs attached to the core proteins are chondroitin sulfate and keratan sulfate.

The proteoglycans themselves are attached, by special linker proteins to long, rigid molecules of hyaluronic acid (HA). HA itself is a GAG, but is composed of several thousand disaccharide units, rather than several hundred or less, as are other GAGs. About eighty proteoglycans are attached to one molecule of HA. The repeating units of chondroitin sulfate are D-glucuronic acid and N-acetylgalactosamine-(4 or 6)-sulfate. The repeating units of keratan sulfate are galactose or galactose 6-sulfate and N-acetylglucosamine 6-sulfate. The repeating units of hyaluronic acid are D-glucuronic acid and N-acetylglucosamine. The properties of proteoglycans in cartilage can be summarized as follows:

- *Basic unit:* glycosaminoglycans (GAGs) which are mutually repelled between neighboring GAGs.
- *Proteoglycan:* hyaluronic acid, link protein, the GAG chains: 200-400 nm in length.

- *Chondroitin sulfate chains (CS)*: decrease with aging.
- *Keratan sulfate chains (KS)*: increase with development and aging.

The matrix also has collagen fibers, but these are of a finer nature (collagen Type II vs. collagen Type I) than the collagen fibers in most other connective tissues. The macromolecules are bound to the thin collagen fibres by electrostatic interactions and cross-linking glycoproteins. Properties of collagen fibers in articular cartilage:

- biological unit: tropocollagen
- mechanical properties: tensile stiffness and strength
- distribution of collagen in articular cartilage (Fig. 1.14)
 - *superficial tangential zone*: parallel to the articular surface.
 - *middle zone*: randomly distributed.
 - *deep zone*: perpendicular to cartilage-calcified cartilage interface.

Between 60 and 80 percent of the net weight of cartilage is water, and this large component of water accounts for the resilient nature of cartilage. Water is attracted to the negative charges in the abundant sulfate and carboxyl groups on the GAGs. This hydration permits diffusion of water-soluble molecules in the ground substance. However the movement of large molecules and bacteria is inhibited. Cartilage is poorly vascularized, and gets most of its nutrients through diffusion. In the adult, repair is poor.

There are three kinds of cartilage, hyaline cartilage, elastic cartilage and fibrocartilage. Hyaline cartilage is the most abundant type of cartilage. Most of the skeleton of the fetus is laid down in cartilage before being replaced by bone. Hyaline cartilage in the adult is found in the nose, parts of the respiratory tract, at the ends of ribs and at the articular surfaces of bones. Fine collagenous fibers are scattered throughout the ground substance, but they are not ordinarily visible in H and E preparations. Thus, the intercellular substance (matrix) appears relatively homogeneous. The cartilage cells are called chondrocytes and lie within little spaces, the lacunae. Cells lying within the lacunae are found only in cartilage and bone. Hyaline cartilage, with the exception of that associated with joints (articular cartilage), is surrounded by a dense connective tissue capsule, the perichondrium. The structure of elastic cartilage is very similar to that of hyaline cartilage, but in addition to the other components, its matrix has elastic fibres and

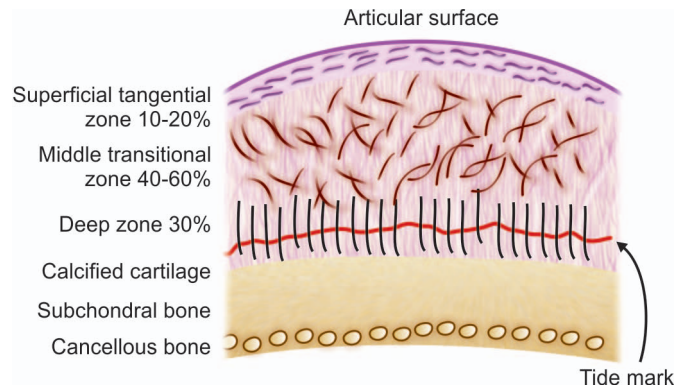


Fig. 1.14: Arrangement of collagen in cartilage

interconnecting sheets of elastic material. This gives elastic cartilage an elasticity which is not present in hyaline cartilage. Elastic cartilage is found in the external ear, the walls of the external auditory canal, the Eustachian tube, the epiglottis and the larynx. Fibrocartilage has characteristics intermediate between those of hyaline cartilage and dense connective tissue. Its presence indicates the need for resistance to compression and shear forces. It is found in the intervertebral disks, the symphysis pubis, the articular discs of the sternoclavicular and temporomandibular joints, the menisci of the knee joint and some places where ligaments or tendons attach to bones. The amount of cartilage in fibrocartilage is variable, it generally occupies a smaller amount of the tissue and there is no perichondrium.

Perichondrium has 2 layers:

- *External*—fibrous; made of dense irregular connective tissue.
- *Internal*—cellular (chondrogenic); contains many osteoblasts and blood vessels.

Functions: cartilage nutrition, appositional growth, and regeneration.

Cells

- **Chondroblasts**—less differentiated cartilage cells, originate from non-differentiated mesenchyme; have a flattened shape; a well-developed rough endoplasmic reticulum in a basophilic cytoplasm; *function*—elaboration of cartilage intercellular matter; under certain circumstances chondroblasts are capable of producing matrix-degrading enzymes - collagenase, elastase, hyaluronidase; *reside* in the

internal layer of periosteum and in the depth of matrix—within lacunae; chondroblasts mature into chondrocytes.

- Chondrocytes—differentiated cartilage cells; of round or angular shapes, with advancing cellular age chondrocytes progressively lose their rough endoplasmic reticulum; *function*—elaboration of cartilage intercellular matter; under certain circumstances chondroblasts are capable of producing matrix-degrading enzymes—collagenase, elastase, hyaluronidase; *reside* in the depth of matrix—within minute special cavities, lacunae; sometimes the number of cartilage cells in one lacunae is more than one, it is the consequence of cell division; quite often the division is accomplished through amitosis; such cellular groups are called isogenic groups.

Lubrication Mechanism of Cartilages

There are two types of lubrication mechanisms involved namely, the boundary lubrication and the fluid film lubrication. The *boundary lubrication* depends upon the chemical adsorption of a monolayer of lubricant molecules onto the articular surfaces and also depends on the chemical property of lubricants.

Fluid film lubrication (Fig. 1.15): In this type, a much thicker film of lubricant causes a relatively large separation of the two bearing surfaces. Elastohydrodynamic fluid films of both the sliding and the squeeze type probably play an important role in lubricating the joint. With high load and low speeds of relative motion, the fluid film will decrease in thickness as the fluid is squeezed out from between the surfaces. Under very high loading conditions, the fluid film may be eliminated, allowing surface-to-surface contact.

Cartilage Replacement Mechanisms

Growth Plate

The epiphyseal growth plate is made up of three types of tissues: the cartilage component divided into distinct zones, the bony tissue of the metaphysis and the fibrous tissue that surrounds the growth plate. The vascular supply to the growth plate is illustrated in Figure 1.16. The secondary ossification centre is supplied by the epiphyseal artery, branches of which end in the proliferating cartilage zone. The metaphysis is supplied

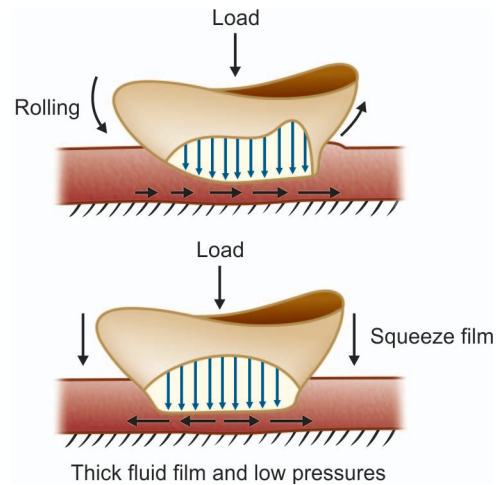


Fig. 1.15: Schematic diagram for fluid film lubrication

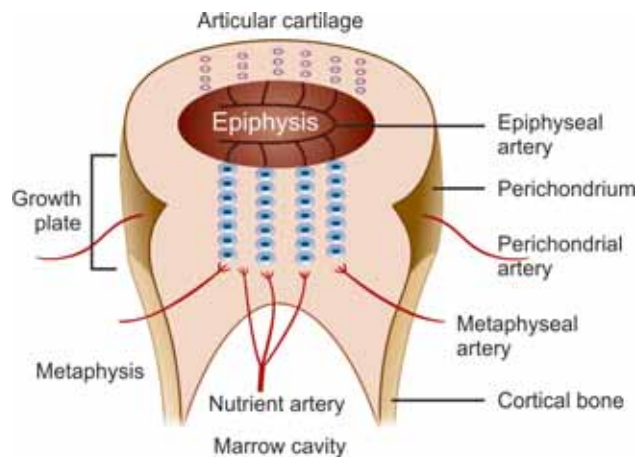


Fig. 1.16: Blood supply of epiphysis

mainly by the nutrient artery, with the periphery having an additional supply from metaphyseal vessels. Terminal branches of these arteries end in capillary loops below intact cartilage septae that delineate the end of the cartilage zone. These capillaries drain into the large central vein of the diaphysis. Since there are no branches from metaphyseal or epiphyseal arteries to the hypertrophic zone, this region of the growth plate is avascular. Only the proliferative zone has an abundant blood supply.

The cartilage matrix is primarily composed of collagen and proteoglycans. These macromolecules play a critical role in the development and maintenance of a variety

of functions including tissue strength, architecture, and cell to cell interactions. If abnormal molecules are present in the matrix, it can lose its functional integrity, lose the organized arrangement of chondrocytes and their closely regulated proliferation and biosynthesis will also be disrupted. Such abnormalities are called dyschondroplasias, and affected individuals suffer from dwarfism. Fortunately, the understanding of cartilage matrix molecules has progressed significantly in recent years with the development of techniques enabling improved protein characterization and localization, together with the knowledge of the gene structure of many matrix molecules. It is now known that genetic defects of a single matrix molecule are the cause of some of these dyschondroplasias.

Type II collagen is the most abundant of the collagens in the growth plate, and since it is found almost exclusively in cartilage, it is a specific phenotype marker for chondrocytes. Type II collagen is composed of three identical chains that are wound into the characteristic triple helix of the collagen molecule. Type II collagen molecules form banded fibres seen with the electron microscope and are therefore classified as fibre forming (class I) collagen. In the developing limb and in models of endochondral ossification, type II collagen synthesis can be correlated with chondrogenesis. Type II procollagen may be expressed in two forms, IIA or IIB, due to differential splicing of recently transcribed mRNA. In embryonic human vertebral column, type IIB mRNA expression is correlated with cartilage matrix synthesis, whereas IIA is expressed in pre-chondrocytes, the cells surrounding the cartilage. Type XI collagen, also a class I collagen, is present in cartilage matrix and is integrated into the interior of type II collagen fibrils. Its function is not known. Type IX collagen is also found in cartilage, but is not a fibre forming collagen since it will not form supramolecular aggregates alone. Type IX is associated with the exterior of the type II collagen molecules and since it has a single glycosaminoglycan side chain, it is also a proteoglycan.

Type X collagen is a short chain, non-fibril forming collagen with a restricted tissue distribution within the hypertrophic calcifying region of growth plates in fetal and developing bone, where it makes up 45 percent of total collagen. It has been proposed that type X collagen may play a role in regulating mineralization of cartilage calcification, however, this remains to be proven.

The other main structural component of cartilage is proteoglycan. Proteoglycans are proteins with one or more attached glycosaminoglycan side chains, e.g. chondroitin sulphate, heparan sulphate, dermatan sulphate. These sulphated side chains occupy approximately two thirds of the C terminus region of the molecule, while the other third, the carbohydrate-rich portion, binds to hyaluronic acid. The main proteoglycan of cartilage is aggrecan, a large proteoglycan composed of approximately 90 percent chondroitin sulphate chains. Aggrecan is found as multi-molecular aggregates composed of many proteoglycan monomers (up to 100) bound to hyaluronan. A small link protein helps to stabilize the aggregate. Synthesis of aggrecan is another specific marker of the chondrocyte phenotype.

Another important matrix component is the enzyme alkaline phosphatase (ALP). ALP is abundant in matrix vesicles and on the plasma membrane of the maturing chondrocytes, and is required in the calcification process although the precise mechanism of action remains unclear. Growth plate chondrocytes are organized into different zones (Fig. 1.17) with each cell population being part of a different stage of maturation in the endochondral sequence. Zone I has otherwise been described as the reserve or resting zone. Cells exist singly or in pairs separated by an abundant extracellular matrix, and have low rates of proliferation. Proteoglycan synthesis and type IIB collagen synthesis is low. However, these cells have a high lipid body and vacuole content that has led to the suggestion that this zone is involved with storage for later nutritional requirements. The adoption of the term 'reserve zone' to describe this region may be inappropriate because these cells do not transcribe type IIA collagen, the marker of pre-chondrocytic cells, evidence that the cells have already differentiated into chondrocytes, i.e. this is not a germinal layer of 'mother cartilage cells'.

Zone II is otherwise described as the upper proliferative or columnar region. The function of the proliferative zones is matrix production and cell division that result in longitudinal growth. Chondrocytes assume a flattened appearance and are arranged in longitudinal columns. The zone is the true germinal layer of the growth plate, with cells actively dividing. Type II collagen synthesis and mRNA expression increase in this zone, as does that of type XI and aggrecan, although in bovine growth plate type IIB collagen levels are relatively higher.

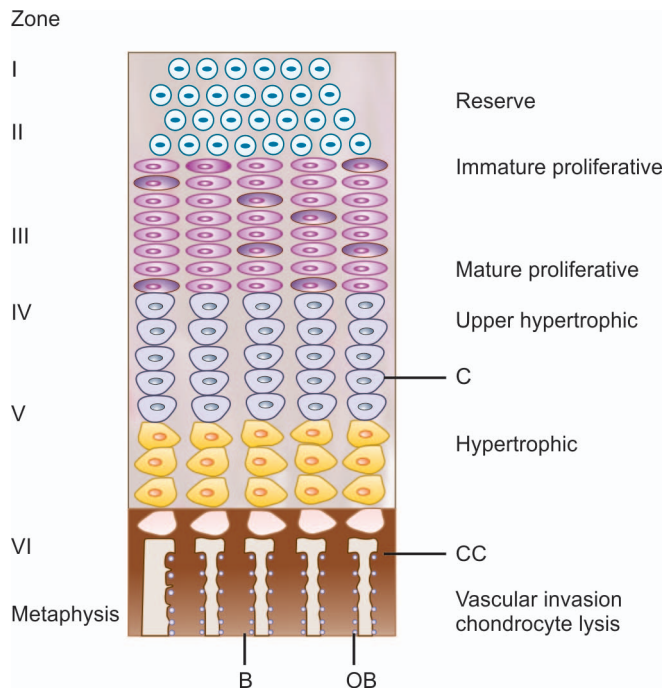


Fig. 1.17: Schematic diagram of a longitudinal section through the epiphyseal growth plate. B = bone, OB = osteoblast, CC = calcified cartilage, C = cartilage matrix

Cells of zone III, the lower, more mature region of the proliferating zone, are morphologically no different from those of zone II, but have decreased DNA synthesis. Type II collagen synthesis remains high; studies of human fetal growth plate report the highest levels of mRNA for type II collagen in these cells (Sandberg et al, 1988).

Zone IV is the upper hypertrophic zone, where cell size abruptly increases and the columnar arrangement is less regular. Although not proliferating, hypertrophic zone cells retain the full complement of cytoplasmic components, and light microscopy reveals increasing vacuolation of the cells. Hypertrophic chondrocytes are metabolically active cells, with overall matrix synthesis per cell increased approximately three-fold, compared to the proliferative zone. The main matrix components synthesized are types II and X collagen and aggrecan.

Zone V is the zone of the terminal chondrocyte. The end of this zone is marked by the last intact transverse cartilage septum. Matrix calcification occurs in longitudinal septae between the columns of chondrocytes, and this calcified matrix becomes the scaffolding for bone deposition in the metaphysis. The hypertrophic zone contains the highest levels of alkaline phosphatase. The

traditional view was that these cells were metabolically very inactive, and that increasing vacuolation indicated death by hypoxia. However, these cells are clearly actively involved in the synthesis of type X and type II collagen. Improvements in techniques of growth plate fixation that retain chondrocyte morphology have led to the proposal that a terminal chondrocyte spends most of its life as a fully viable cell indistinguishable from hypertrophic chondrocytes positioned further proximally in the growth plate. The cells then die by apoptosis, a distinct biological form of cell death, lasting approximately 18 percent of a terminal chondrocyte's lifespan. Apoptosis may be triggered by the metaphyseal vasculature beyond the last intact cartilage septum.

Zone VI is the junction of the growth plate with the metaphysis, the region where the transition from cartilage to bone occurs. Chondrocyte lysis is evident from empty lacunae invaded by vascular endothelial loops. The vascular region of calcified cartilage is the primary spongiosum, upon which osteoblasts lay down unmineralized bone, the osteoid. Metaphyseal bone formation is associated with type I procollagen mRNA expression in the empty lacunae, osteoid, bone and perichondrium. Type I collagen, a marker of the osteoblast phenotype, is immunolocalized to the same areas, while types II and X collagen have restricted immunolocalization to calcified cartilage trabecular remnants within spongy bone. Newly formed woven metaphyseal bone is gradually replaced by lamellar bone following osteoclastic degradation of bony matrix and chondroclastic removal of remaining cartilage trabeculae. At the same time, external reshaping of the bone is brought about by surface osteoclastic bone resorption and appositional bone formation by periosteally derived osteoblasts.

Synchondrosis

The structure in the cranial base which resembles the growth plates are synchondroses. A synchondrosis is a type of immovable joint in which the articulating structures are joined together by hyaline cartilage. Synchondroses are formed between the epiphyses (ends) and diaphyses (shafts) of long bones (Fig. 1.18). It includes the numerous temporary cartilaginous junctions between diaphysis and epiphysis in the immature post cranial skeleton and also in the regions of unossified cartilage between skull components developing in the

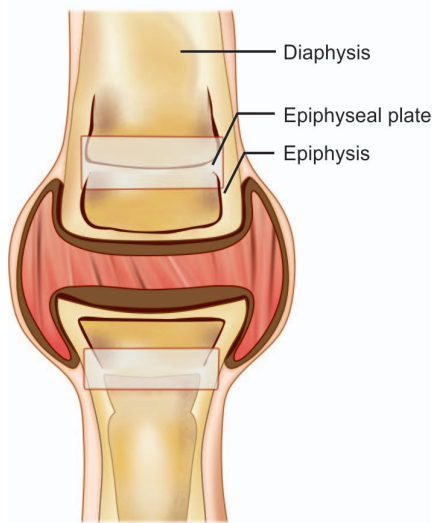


Fig. 1.18: Diagrammatic representation of synchondrosis

chondrocranium (e.g. between the sphenoid and occipital bones). The most important synchondrosis in the growth of craniofacial region is spheno-occipital synchondrosis. The sphenoid bone consists of a central body and lateral greater and lesser wings. The body is divided embryologically into a presphenoid segment and the post sphenoid segments. The prephenoid segment lies anterior to tuberculum sellae and post sphenoid segment comprises the sella turcica and dorsum sellae. These two parts fuse by eight months of fetal life. Posterior to the post sphenoid, the cartilage of the basilar part of the occipital bone becomes ossified simultaneously. Both the postsphenoid and basilar occipitalis continue ossification until all that remains is a plate of cartilage between them, called the *spheno-occipital synchondrosis*. Growth of spheno-occipital synchondrosis is responsible for lengthening between foramen magnum and sella turcica. This continues up to latter half of second decade of life. The cellular arrangement of synchondroses suggests that it looks like butting together of two growth plates with reserve cartilage layers back to back. As a result of this arrangement, interstitial expansion is bidirectional, increasing the size of the bones in both sides simultaneously (Fig. 1.19).

Nasal Cartilage (Fig. 1.20)

Nasal cartilage is a thin cartilaginous plate located between vomer, perpendicular plate of the ethmoid and nasal

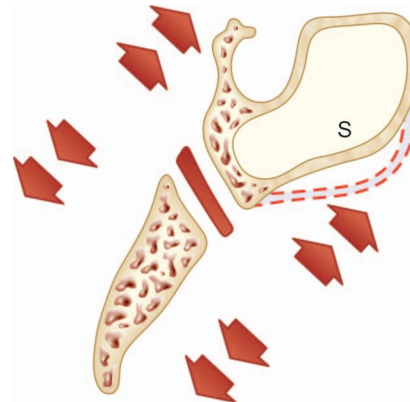


Fig. 1.19: Bidirectional interstitial growth of synchondrosis

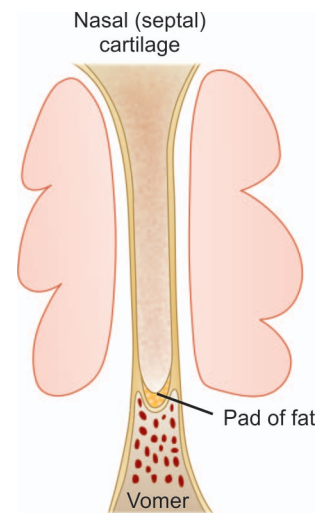


Fig. 1.20: Cross-section of nasal cartilage

bone. Histological examinations reveal that there is endochondral ossification taking place at the septo-ethmoidal junction and that there is an area of proliferation at the vomeral edge of the cartilage. In the palatal area, there is resorption on the nasal side and apposition on the oral side of the bony palate. These findings appear to support the general opinion. The role of the cartilaginous nasal septum has been discussed by many authors also and the general consensus seems to be that it provides a thrusting force which carries the maxilla forward and downward during growth.

In prenatal life, nasal septum cartilage lies behind the cranial base cartilages. In front and below it is

attached to premaxillary bone. The lower edge is attached to vomer. Posteriorly it merges with mesethmoid cartilage.

Condylar Cartilage

The condylar cartilage is a secondary type of cartilage which was transformed phylogenetically from the periosteum. Secondary (accessory or embryonic) cartilages are local mesenchymal cartilage formations, primarily associated with membrane bones or with fracture of long bones; ontogenetically and phylogenetically they do not develop from the primary cartilaginous skeleton. Apart from the embryonic origin, the condylar cartilage differs from primary cartilages (such as the epiphyseal growth plates and the synchondroses) in growth pattern, in histologic organization, and in antigenicity.

This cartilage is a latecomer, a secondary cartilage, and not a part of the Meckel's cartilage that acts as the model for the early development of the mandible. It is not an articular cartilage, nor is it an epiphyseal growth plate. It does not even form from the same embryonic precursor tissue as the epiphyseal cartilages, a fact which may have something to do with its structure and function. It is claimed that the condylar cartilage grows not interstitially, like the epiphyseal cartilages, but appositionally from the deepest layer of the connective tissue cover of the condyle. This mitotic layer responsible for the increase of the cartilage is also called the intermediate layer. It is located between the surface of the condyle and the cartilaginous portion of it, and the cells of this layer are not cartilage cells but are rather like undifferentiated mesenchymal cells. In the epiphyseal cartilages, as we know, the proliferating cells are cartilage cells. There are other differences between the condylar cartilage and the epiphyseal growth cartilages. The structural organization present in the epiphyseal growth apparatuses is lacking in the condylar cartilage, and the zone of nonhypertrophic cartilage cells in the condyle is very narrow, the forming cartilage cells turning hypertrophic almost immediately, as in the clavicle. It is of special interest that the whole hypertrophic area in the condylar cartilage seems to be in a state of mineralization, whereas in the epiphyseal growth apparatuses only the degenerative zone is mineralizing. Finally, the so-called primary spongiosa, always present

in the long bones, seems to be absent in the condyle. In regard to the function of the condylar cartilage, differences have been found to exist between it and the epiphysis. The condylar cartilage is highly responsive to mechanical stimuli and responds differently from the epiphyseal cartilages to various hormonal and chemical agents. The decisive point is the question of the tissue-separating force or the independent growth-promoting potential. As mentioned earlier, the existence of this force or potential has been implicit in the interpretation of the function of the condylar cartilage in most descriptions of the condyle. This problem can again be tackled by way of transplantation. If the condylar cartilage is transplanted to a relatively nonfunctional site, such as the subcutaneous or brain tissue, it does not maintain its structure and does not behave like the condylar cartilage in situ. Only when it is accompanied by a piece of adjacent bony ramus may the transplant grow, and even then the structure is not maintained in the beautiful manner as observed in transplanted epiphyseal cartilages. Tissue-culture studies have also demonstrated lack of growth of the condylar cartilage.

There are four different zones in the condylar cartilage. The outer dense fibrous connective tissue zone that are sparsely vascular. Then the proliferation zone of undifferentiated connective tissue cells which becomes differentiated to chondroblasts. The hyaline cartilage zone with randomly distributed chondroblasts and hypertrophied cells. The matrix of these cells is more towards the condyle (Fig. 1.21).

The endochondral ossification zone in which the cartilage is resorbed and replaced with trabecular bone. Condylar cartilage can be differentiated from the epiphyseal cartilage in that the outer fibrous covering is only present in condylar cartilage. In the epiphyseal cartilage mineralization starts only below the hypertrophied layer, whereas in condylar cartilage the mineralization starts in the hypertrophied layer. The condylar cartilage does not have a matrix surrounding its cells in contrast to the epiphyseal cartilage.

Primary and Secondary Cartilages

The features of primary cartilages are:

- They are derivatives of primordial cartilage.
- In primary cartilage, chondroblasts divide and synthesize intercellular matrix.

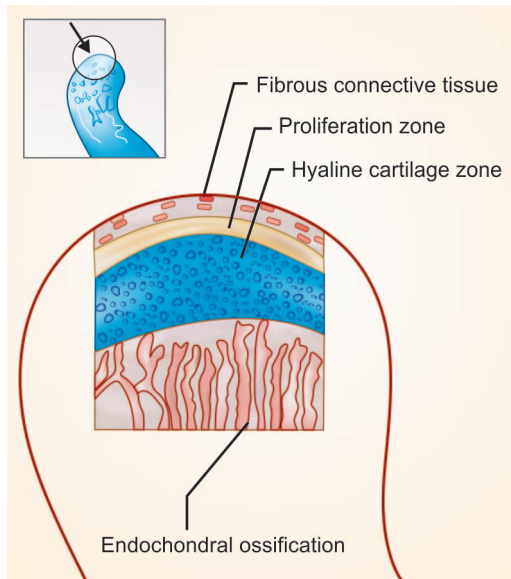


Fig. 1.21: Structure of condyle

- The dividing chondroblasts are surrounded by cartilaginous matrix.
 - Cells arranged in columnar fashion
 - Since surrounded by cartilaginous matrix, primary cartilage is not influenced by local environmental factors, e.g. Epiphyseal cartilages, synchondroses
 - Growth is interstitial. Hence 3 dimensional growth
 - Considered to be a genetic pacemaker for growth.
- Features of secondary cartilages include:

- Secondary cartilage forms on a membranous bone
- No intercellular matrix
- Not surrounded by cartilaginous matrix
- Cells are arranged in haphazard manner
- Affected by external influences which will stimulate growth of cartilage, e.g. condylar cartilage
- Only peripheral growth takes place
- Contributes only to regional adaptive growth.

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Chapter

2

Physiology of Bone

CHAPTER OUTLINE

- Bone Turnover
- Modeling and Remodeling
- Basic Multicellular Unit (BMU)
- Mechanical Influence on Bone
- Bone Regulators
- Purpose of Bone Remodeling
- Goals of Remodeling
- Remodeling Process
- Reactions of Bone
- Bone Assessment Methodologies

Bone is a complex, living tissue that is constantly adapting to metabolic and structural demands. Because it is a mineralized tissue, all changes in external osseous form occur along vascularised periosteal surfaces via uncoupled anabolic and catabolic modeling events. The ability of a bone to function effectively under the loads that are imposed on it depends upon two factors: the properties of the bone material, and arrangement of this material in space—the size and shape of the bone. Eugene Roberts referred orthodontists as craniofacial bone specialists and hence a thorough knowledge about bone physiology will help the orthodontist to deal with the patients more effectively and efficiently.

BONE TURNOVER

Mature bone undergoes a continuous process of resorption and formation known as remodeling. Osteoclasts remove old bone by acidification and proteolytic digestion. Later, osteoblasts migrate to this area, and deposit osteoid, into the cavity. The collagen then becomes infused with calcium phosphate mineral. Remodeling provides a mechanism whereby the skeleton

can adapt to mechanical, metabolic, and hormonal stimuli or stresses. For many years, researchers have tried to understand the mechanical influences on living bone.

MODELING AND REMODELING (FIG. 2.1)

The physiologic concept of bone remodeling (turnover) is largely attributed to important biologic activities like modeling and remodeling. Most experiments and theory about bone adaptation are concerned with the placing or replacing of bony mass. This is usually termed 'modeling' and is produced by the probably rather

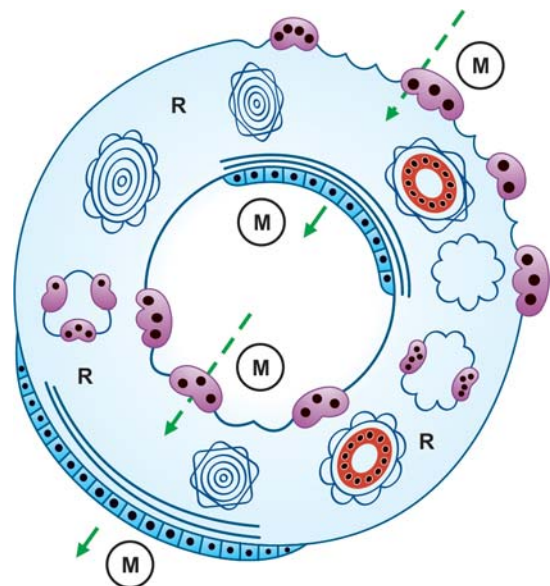


Fig. 2.1: Schematic diagram illustrates the integration of anabolic and catabolic modeling activity (M) along bone surfaces with internal remodeling (R) (turnover) to produce new secondary osteons

uncoordinated activity of bone cells. It should be distinguished from 'remodeling' which is also a matter of lively concern, particularly where it occurs in cancellous bone, in which osteoclasts and osteoblasts work together in a coordinated sequence to replace bone and usually leave the total amount of bone unaltered, in the form of secondary osteons (Haversian systems).

The modern physiologic concept of bone remodeling (turnover) is largely attributed to Frost. Harold Frost, (1922-2004) is considered to be the father of the modern concepts of bone physiology for his extensive work. Frost differentiates bone "modeling" from "remodeling". *Bone modeling* is a mechanically mediated adaptive process for changing a bone's size, shape, or position. Bone modeling is an uncoupled process, meaning anabolic and catabolic sites are controlled independently. Bone modeling, an important element of skeletal growth, functions as a lifelong optimization process for adapting bone mass and architecture to functional needs. Modeling also called as macro modeling by some authors is an activity primarily found during growth and is responsible for the final shape of the bones. *Bone Remodeling* is the physiologic term for internal turnover of a mineralized tissue, without a change in its overall form. It is a coupled sequence of catabolic (resorptive) and anabolic (osteogenic) events to support calcium homeostasis and repair (renew) aged or damaged mineralized tissue. Both modeling and remodeling are the result of the controlled activity of osteoblasts and osteoclasts. The difference is in modeling, both these two cells act over a large surface area, removing or forming large volumes of bone mass which is active during growth period. Remodeling on the other hand is active throughout life and serves to modify shape of skeleton, architecture, bone volume and to repair microdamage.

BASIC MULTICELLULAR UNIT (BMU)

Bone replacement either by modeling or remodeling is initiated by osteoclastic resorption followed soon after by osteoblastic formation. These are commonly regarded as independent processes, but in reality resorption and formation are closely linked within discrete temporary anatomic structures, first described by Frost who gave them the name "basic metabolizing units", a term he later changed to "basic multicellular units", usually abbreviated BMU. The individuality of the BMU is most

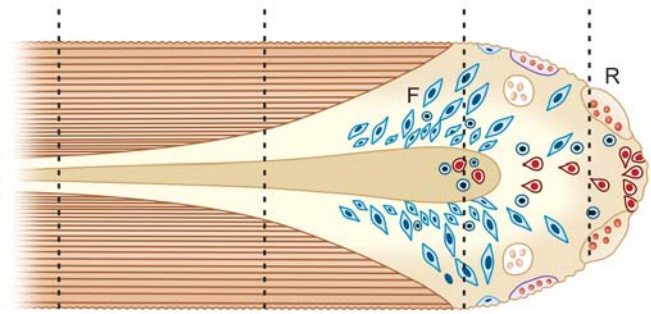


Fig. 2.2: A schematic illustration, of a longitudinal section through a cutting/filling cone in cortical bone, illustrates the perivascular cellular activity associated with the coupled resorption (R) and formation (F) responses

readily demonstrated in cortical bone but there is compelling evidence for the same concept in cancellous bone. Parfit (1994) states that a fully developed BMU consists of a team of osteoclasts in front forming the cutting cone or hemicone, a team of osteoblasts behind forming the closing cone or hemicone, some form of blood supply, and associated connective tissue (Fig. 2.2). The BMU exists and moves in three dimensions, excavating and refilling a tunnel through cortical bone or a trench across the surface of cancellous bone. A cortical BMU travels for about 4000 μm at about 20 $\mu\text{m}/\text{day}$, taking about 200 days. A cancellous BMU travels about half this distance at about half the speed, taking about the same period of time. While moving through or across the surface of bone, the cellular components of a BMU maintain the same spatial and temporal relationships to each other.

MECHANICAL INFLUENCE ON BONE

It is generally accepted that mechanical load plays an important role in maintenance and adaptation of the skeleton. Bone mass and bone architecture are believed to be adapted to the external loading conditions. Wolff in 1892 put forward the famous law of transformation of bone which states that *Every change in the form and function of bone or of their function alone is followed by certain definite changes in their internal architecture and equally definite alteration in their external conformation, in accordance with mathematical laws.* This paved way for many pioneers like Sicher, Frost, to do extensive research in bone physiology.

Current thinking about Wolff's law revolves around several key concepts and observations. First, it is postulated that bone contains sensor cells that monitor mechanical strain (or another load-related variable), compare it to a physiologically desirable range of values, and activate corrective biological processes when the sensed variable falls outside this range. Second, many investigators have suggested that osteocytes, distributed throughout the bone matrix, are bone's mechanosensing cells (Cowin, 1995). There has been considerable speculation that osteocytes produce a signal proportional to mechanical loading by sensing strain on bone surfaces through stretch-activated ion channels, flow of interstitial fluid (Weinman and Cowin, 1994), electrical potentials, or some other phenomenon. Adaptation to mechanical load requires the existence of mechanosensors in bone. This role is believed to be played by osteocytes (Lanyon, 1987). According to present opinion, osteocytes transduce mechanical signals into bone-formative stimuli, transported through the osteocytic canalicular network (Burger and Klein-Nulend, 1999). It is assumed that these recruit and activate osteoblasts to form bone, where and while the stimulus exceeds a certain threshold value. Hence, supernormal osteocyte signaling from increased bone strains would stimulate osteoblast formation. It is thought that the stimulus for osteoclasts to resorb bone is related to mechanical disuse or to microcracks within the bone matrix, and probably both. Equally strong is the case for microdamage in the bone-tissue matrix as a controlling factor for osteoclast resorption (Burger and Klein-Nulend, 1999; Verborgt et al., 2000). Verborgt et al. (2000) showed strong associations between microdamage, osteocyte apoptosis, and subsequent bone resorption. This suggests that the signaling mechanisms for osteoclastic removal of microdamage might be related to osteocyte apoptosis. Burger and Klein-Nulend (1999) suggested that microdamage of bone interferes with osteocyte signaling through the canalicular network. As a result, the osteocytes would no longer suppress osteoclast recruitment.

Third, it has been suggested that osteocytes also sense fatigue damage and transmit signals activating remodeling to remove the damage. The work of Bentolila, et al has shown that microdamage in cortical bone is associated with increased activation of remodeling, and it is generally assumed that the same is true in cancellous bone. This remodeling is postulated

to remove damaged bone and prevent the occurrence of fatigue fracture. The dynamic forces of daily living are known to produce microcracks. As the trabecular bone structure is a mechanically optimal one, implying that all material is frequently stressed in daily life (Van Rietbergen et al., 1999), and the strength of the mineralized tissue is non-homogeneously distributed (Choi and Goldstein, 1992), it is likely that microcracks or diffused damage can occur anywhere, at any time, for a normally functioning individual. Bone formation and bone resorption influence the local trabecular morphology, which changes the local distribution of strain energy density (SED) rate, alternative signaling activation of osteocytes, and so on. The formation of resorption cavities produces local stress raisers for the remaining bone tissue, which subsequently stimulates bone formation, in accordance with the strain-rate dependent metabolic rule assumed. A fourth key concept, developed from the seminal work of Rodan and Martin, suggests that cells of the osteoblast lineage control the initiation of remodeling. Subsequently, many investigators have adopted the modified hypothesis that those "retired" osteoblasts, known as bone lining cells, are responsible for activating BMUs to remodel bone in response to signals from osteocytes or hormones. Taken together, these four concepts form an attractive model in which osteocytes sense mechanical changes and initiate remodeling to modify bone structure accordingly.

The differential response to mechanical influence is evident in maxilla and mandible. The cortices of maxilla are relatively thinner when compared to mandible and they are interconnected by a network of trabeculae. On the other hand, mandible has thick cortex and the trabeculae are radially oriented. Maxilla resembles that of body of vertebrae. The reason for such arrangement in maxilla could be because (i) the forces from maxilla are transferred to cranium and (ii) maxilla is loaded predominantly in compression. In the case of mandible, the forces are not transferred and mandible has to bear the entire load. Also mandible is loaded in both bending and torsion.

BONE REGULATORS

The regulators of bone are classified as:

- Endocrine regulators.
- Paracrine regulators (produced locally and active in the immediate vicinity).

- Autocrine regulators (Produced intracellularly).
- Neurotransmitters.

The important endocrine regulators are parathyroid hormone, calcitonin, vitamin D, vitamin A, estrogens, androgens and growth hormones. PTH acts on bone, intestine and kidney. It enhances calcium resorption in intestine and kidneys. Parathyroid hormone increases the flow of calcium into the calcium pool and maintains the body's extracellular calcium levels at a relatively constant level. Osteoblasts are the only bone cells that have parathyroid hormone receptors. This hormone can induce cytoskeletal changes in osteoblasts. Experimental studies have shown that PTH leads to bone apposition. Calcitonin acts on osteoclasts and stops resorption of bone. Calcitonin is produced by thyroid C cells. Vitamin D is involved in mineralization of bone. Vitamin A excess, decreases the formation of bone and it interacts with vitamin D metabolism and influence PTH activity and production. Estrogen deficiency in females tend to produce bone loss whereas androgens contribute to the formation and maintenance of bone mass.

Bone metabolism is also affected by a series of proteins, or growth factors, released from platelets, macrophages, and fibroblasts. These proteins cause healing bone to vascularize, solidify, incorporate, and function mechanically. They can induce mesenchymal-derived cells, such as monocytes and fibroblasts, to migrate, proliferate, and differentiate into bone cells. The proteins that enhance bone healing include the BMPs, insulin-like growth factors, transforming growth factors, platelet derived growth factor, and fibroblast growth factor among others. The most well known of these proteins are the Bone morphogenetic proteins (BMPs), a family of glycoproteins derived from bone matrix. Bone morphogenetic proteins were first postulated in 1952. Since then nearly seven related proteins have been extracted from bone. Bone morphogenetic proteins induce mesenchymal cells to differentiate into bone cells. Other proteins influence bone healing in different ways. *Transforming growth factor*—regulates angiogenesis, bone formation, extracellular matrix synthesis, and controls cell-mediated activities. Osteonectin, fibronectin, and osteocalcin promote cell attachment, facilitate cell migration, and activate cells. Insulin-like growth factors (IGFs) are among the most abundant growth factors present in bone. *In vitro*, bone-derived cells both

produce and respond to IGFs I and II, suggesting that these growth factors play an autocrine role in the regulation of bone turnover. The novel TNF superfamily members RANKL and OPG (osteoprogenin) are essential paracrine mediators of bone metabolism and immune functions. Neurotransmitter regulation of bone metabolism has been a topic of increasing clinical interest and investigation. Collectively, anatomical and *in vitro* studies suggest that bone metabolism may be influenced by the nervous system; for example, bone and periosteum have been shown to be innervated by both sympathetic and sensory nerves. Anatomical studies of nerve terminals innervating bone have revealed the presence of several neuropeptides, including calcitonin gene related peptide (CGRP), vasoactive intestinal peptide, substance P, and neuropeptide Y27; glutamate-containing terminals have also been described in a dense and intimate network in bone tissue. Fann et al. demonstrated that bone morphogenetic proteins (BMP-2 and BMP-6) induce mRNAs for some neuropeptide and neurotransmitter synthetic enzymes *in vitro*. Vasoactive intestinal polypeptide (VIP) was shown to stimulate prostaglandin (PGE2) and cyclic AMP production in human osteoblast-like cells.

PURPOSE OF BONE REMODELING

Bone remodeling is the mechanism of bone replacement in the vertebrate skeleton. Considering the reasons for replacement, it must be required to preserve the functional capacity of bone, which is in some way compromised if it is allowed to become too old. The primary function of bone is mechanical load bearing, which is carried out by cortical bone throughout the skeleton and by peripheral cancellous bone. Subsidiary functions are to participate in plasma calcium homeostasis and to support hematopoiesis, which are carried out mainly by central cancellous bone. The remodeling apparatus can accommodate circadian fluctuation in calcium balance and supply a temporary need for additional calcium lasting for a few months, and accomplishes slow thickening of trabeculae during growth as well as slow elimination of surplus bone in response to the age-related decline in physical activity. However, the main purpose of remodeling is to prevent degradation of function as bone becomes older.

GOALS OF REMODELING

Burr (2002) has outlined three important goals of remodeling. First, it provides a way for the body to alter the balance of essential minerals by increasing or decreasing the concentration of these in serum. Second, it provides a mechanism for the skeleton to adapt to its mechanical environment, reducing the risk for fracture and increasing the organism's chances for passing its genes to the next generation. Third, it provides a mechanism to repair the damage created in bone by repetitive cycles of mechanical loading.

REMODELING PROCESS

Frost has formulated a hypothesis regarding the operative and control mechanisms of the bone during remodeling. This hypothesis explains the organization of skeleton in a hierarchical pattern. Each level of the organization is interdependent with one above. The levels in descending order are as follows:

The body	: L_{IS} (intact subject)
The integrated musculoskeletal system	: L_{SK}
The skeletal organs	: L_O
Three tissue levels	: L_3, L_2, L_1
Units of the musculoskeletal system	
The organized tissue of those units	
The elemental tissues of the system	
Cells	: L_C
Organelles	: L_{org}
Molecular activity	: L_m

The integrated musculoskeletal system is a reference to the articulated skeleton, with its joints, neuromuscular apparatus. Skeletal organs refer to individual bones, joints. The three tissues are designated as upper, middle and lower levels. L_3 or upper intermediary organization include the complete structural units of the organs like epiphysis, physis, metaphysis and bone envelopes (periosteum, endosteum trabeculae). Middle intermediary organization or L_2 includes the tissues of L_3 units; e.g. synovium, growth cartilage, primary and secondary spongiosa, trabecular bone, compact bone etc. Lower intermediary organization or L_1 includes lamellar bone, woven bone, hyaline cartilage, elastic cartilage, etc. Cells refers to the cells of bone, organelles are the membrane bound compartments of the cell like mitochondria, smooth and rough endoplasmic reticulum, lysosomes,

endosomes, nucleus, cytosol and Golgi bodies. Molecules are the chemical entities of the cells and tissues like water, inorganic ions, proteins, RNA, DNA, polysaccharides, lipids, etc.

During adaptation to increased or decreased loading, bone tissue adjusts through a feedback system in which changes in the local mechanical environment, signal bone cells to modify bone structure to the new requirements. This occurs through the processes of bone formation and resorption, with the ultimate result depending upon the balance between osteoblast and osteoclast activity and the anatomical location of these cell types. The rate-limiting factor in orthodontics is the efficiency with which bone is removed in the path of tooth movement. In cortical bone, remodeling mainly occurs via osteonal tunneling, wherein osteoclasts excavate a Haversian canal that is refilled by the action of osteoblasts. These canals are 100-200 μm wide, can be up to 10 mm long, and are oriented along the main loading direction (at an oblique angle to the long axis of the bone). It is important to note, however, that Haversian remodeling does not occur in all animal species. Cancellous bone is remodeled mainly by osteoclasts eroding bone along the surface of the trabeculae, forming Howship's lacunae that can reach depths of 60-70 μm . Osteoblasts follow by refilling the cavity with new bone. One possible mechanism whereby changes in the mechanical environment are transmitted to bone cells is via changes in fluid flow through bone. This could be via one or more of the three levels of porosity that exist within bone tissue. The first is the vascular porosity composed of Volkmann canals and Haversian canals with an average diameter of approximately 10 μm . The second is the lacunar-canalicular porosity around osteocytes, with a mean diameter of 0.1 μm . The third is the collagen hydroxyapatite porosity (which exists between crystals of hydroxyapatite) with a mean diameter of 40 nm. Of the three levels of porosity, the lacunar-canalicular porosity is the primary porosity associated with fluid flow due to mechanical loading. The most distinctive features of bone remodeling of both cortical and trabecular bone is the precise coupling of bone resorption and formation. The ARF process is similar for all types of bone remodeling. The overall duration of the entire ARF process which is the remodeling period is about 151 days. Of which it takes 29 days for resorption and nearly 134 days to refill it by osteoblasts for a size of about

200 to 250 μm in diameter. The bone formation occurs for a longer period of time. The hypothetical sequence of bone remodeling is depicted in Flow chart 2.1.

Reactions of Bone

Bone reactions are in effect, the response of cells in bone to stimuli. If the stimulus is beyond normal range, it leads to pathologic reactions. The various stimuli to which bone reacts can be classified as local or systemic:

<i>Local stimuli</i>	: Mechanical force
	: Agents of other disease process
<i>Generalized stimuli</i>	: Hormonal
	: Nutritional
	: Exogenous substance

The reactions to local group of stimuli results in modeling response of the bone. In orthodontic tooth movement, both resorption and deposition takes place; while in pathologic states either resorption or deposition predominate. The agents of the reaction are the cells namely osteoclasts and osteoblasts acting on surfaces in

bone; fibroblasts, endothelial cells and bone cell precursors acting in marrow and periosteum.

Regional acceleratory phenomenon (RAP): An important reaction of bone which occurs is called regional acceleratory phenomenon, abbreviated to RAP. Frost has explained about RAP. Any regional noxious stimulus can evoke RAP. In this reaction, most of the active vital processes are accelerated: perfusion; growth of bone, cartilage, skin; BMU turnover of woven and lamellar bone; turnover of connective tissues; and enhancement of healing process. When RAP fails to develop, healing is delayed and infection progresses alarmingly. The duration of RAP is in the range of months to years. RAP is highly essential to promote healing.

Bone Assessment Methodologies

The different bone assessment methods are enumerated in Table 2.1.

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Flow chart 2.1: Flow chart depicting the possible sequence of remodeling process

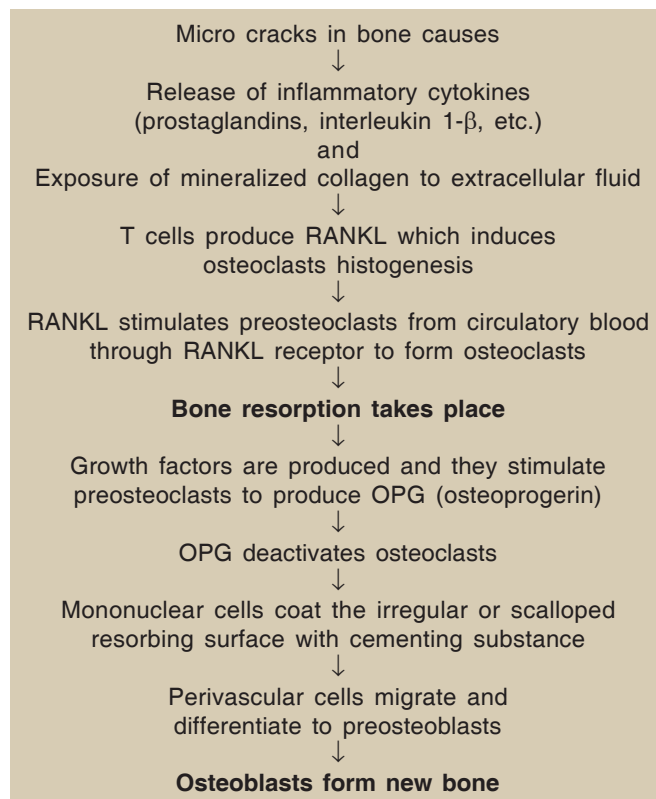


Table 2.1: Different methods used to study bone and their uses

No.	Method	Properties
1.	Mineralized sections	- Both organic and inorganic matrix can be studied simultaneously. - Provide good information about the strength, maturation and turn over rate of cortical bone. - Enhanced cellular details. - But microradiographic analysis is not possible.
2.	Polarized light	- Bone labels quench more rapidly. - Shows the orientation of collagen fibers in bone matrix. - Two specialized types of bone collagen configuration is seen depending upon the loading: (i) Longitudinally aligned collagen fibers which resist tension (ii) Transverse or circumferential collagen fibers which support compression.
3.	Fluorescent labels	- Permanently mark all site of bone mineralization at a specific time. They are called as anabolic markers. - Useful in determining the mechanism of bone growth and functional adaptation. - Example: tetracycline—fluoresce bright yellow; calcein—fluoresce bright green; xylenol—orange fluoresce orange; alizarin complexone—fluoresce red; demeclocycline—fluoresce gold; oxytetracycline—fluoresce greenish yellow. - Multiple fluorochrome method is highly sensitive for assessment of bone growth, healing, functional adaptation and response to loads.
4.	Microradiography	- Mineral density patterns can be assessed in the same sections. - Uses high resolution images.
5.	Microindentation	- Used to determine the material properties of bone. - Developed by Huja, et al.
6.	Back scatter imaging	Is a high resolution method for assessing bone mineral density and surface topography patterns of osseous interface of dental implants.
7.	Microcomputed tomography	- Allows 3 dimensional detection of bone mineral density pattern to a resolution of 5 μm. - Can detect bone remodeling within intact specimen.
8.	Autoradiography	Physiologic index of activity is assessed by quantitative and qualitative assessment of radioactive label uptake.
9.	Nuclear volume morphometry	- In this method, size of the nucleus is measured for assessing the stage of differentiation of osteoblasts precursor cells. - Useful for assessing the mechanism of osteogenesis.
10.	Cell kinetics	- It is a quantitative analysis of cell physiology. - Detects morphologically distinct events in the cell cycle like mitosis, DNA synthesis phase and differentiation, specific change in nuclear volume.
11.	Finite element modeling	It is an analytic engineering technique for measuring the stress and strain in all material including the living tissues.
12.	Microelectrodes	- Detect changes in electrical potential in the tissue due to mechanical loading. - Thin tungsten or glass electrodes are inserted into the periodontal ligament through gingival sulcus in this method. - This method has proved the point that bone is formed in negatively charged or cathode areas and resorption takes place near positively charged or anode areas.

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Chapter 3

Prenatal Craniofacial Growth

CHAPTER OUTLINE

- Period of Ovum
- Period of Embryo
 - Presomite period
 - Gastrulation and neurulation
 - Somite period
 - Branchial arches
 - Development of face
 - Postsomite period
 - Development of tongue
 - Development of palate
 - Development of mandible
 - Neurocranium
 - Cranial base
- Fetal Period
- Development of Temporomandibular Joint (TMJ)
- Changing Relationships in Fetal Face

The organization and complexity of growth and development is clearly evident in the changes that take place in the head and face. Human life starts with the fertilization of ovum by spermatozoa in the fallopian tube

of the female reproductive system. What follows is an incredible cascade of events that culminates in the development of human form. Prenatal craniofacial growth is a highly complex phenomenon with three distinct stages of development. They are:

1. *Period of ovum*: Conception to 7-8 days of intrauterine life [IUL].
2. *Period of embryo*: 2nd to 8th week IUL. This period can be further divided into the following three stages:
 - Presomite 8-20 days
 - Somite 21-31 days
 - Postsomite 4th-8th week.
3. *Period of fetus*: 3rd-10th lunar month.

PERIOD OF OVUM

Fertilization of oocyte by sperm results in the formation of zygote that undergoes rapid mitosis on its passage along the fallopian tube to form a cluster of cells called blastomere (Fig. 3.1). Continuation of mitosis results in a 16 cell stage called morula. The center of morula

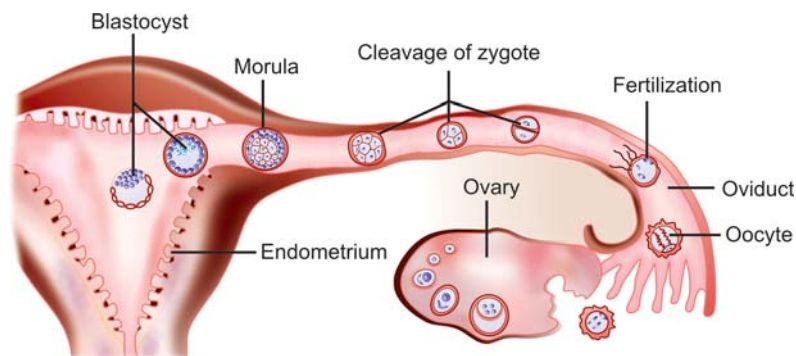
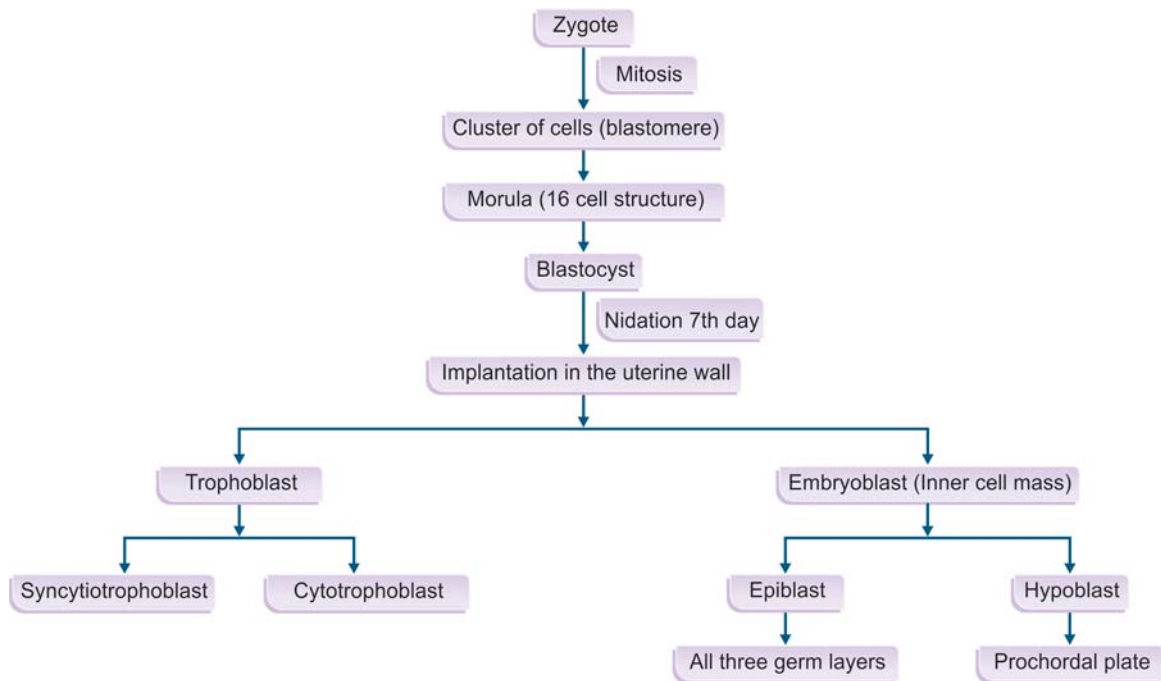


Fig. 3.1: The stages of development of blastocyst as the fertilized ovum traverses through the fallopian tube

Flow chart 3.1: Flow chart depicting the conversion of zygote into germ layers and prochordal plate

cavitates to form a structure called blastocyst. The fluid filled space in the blastocyst is called blastocystic cavity surrounded by a single layer of cells called trophoblastic layer. Inside the blastocyst is the inner cell mass called the embryoblast (Fig. 3.2). The trophoblastic layer forms the embryonic part of the placenta and the inner cell mass develops into the embryo. By the starting of 2nd week of IUL, the blastocyst is implanted into the uterine endometrium (Flow chart 3.1).

PERIOD OF EMBRYO

The period of embryo connotes the development of embryo from the blastocyst. The embryonic period is divided into three stages; presomite, somite and post somite period.

Presomite Period

The presomite period is from the 8th-20th day of intrauterine life. Presomite period is the period of formation of the fetal membranes, amnion and chorion, that provides nutritional supply to the developing embryo and the formation of primary germ layers. After implantation of the blastocyst in the endometrium, trophoblastic layer differentiates into *syncytiotrophoblast*

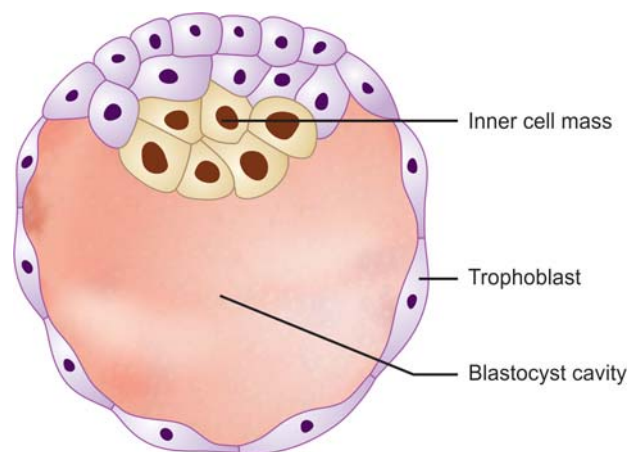


Fig. 3.2: The blastocyst with the embryoblast and trophoblastic layer. Blastocystic cavity is seen within the trophoblastic layer

and *cytotrophoblast* layers. Syncytiotrophoblast, as the name implies is a single multinucleated outer cell that erodes and invades the endometrium and its vessels to establish maternal blood circulation to the developing embryo. The circulation is aptly named *uteroplacental circulation*.

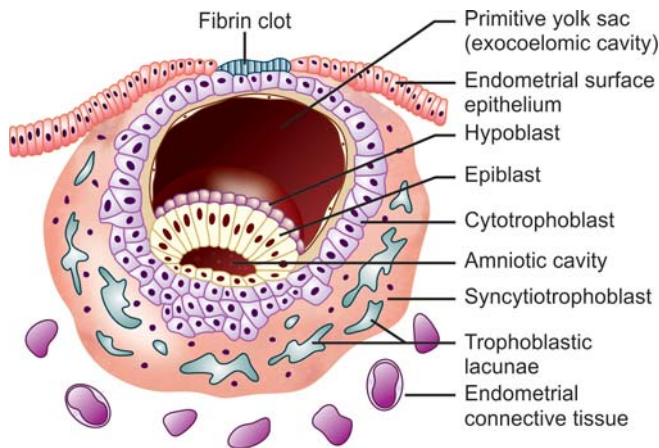


Fig. 3.3: The blastocyst with amniotic cavity developed. Inner cell mass has differentiated into epiblast and hypoblast layers. Syncytiotrophoblast and cytotrophoblast layers are seen. The inner cell mass is a bilaminar disk made of epiblast and hypoblast

The inner cell mass of the blastocyst differentiates into a bilaminar disk, the two layers being *hypoblast*, formed of squamous or cuboidal cells and *epiblast*, formed of columnar cells (Fig. 3.3). Blastocystic cavity, by now called *primitive yolk sac*, is bound partly by

hypoblast and partly by squamous cells. *Amniotic cavity* develops between epiblast and cytotrophoblast. Extra-embryonic mesoderm formed of loose connective tissue, differentiates between the developing embryo and cytotrophoblast. *Chorionic cavity* is formed by fusion of number of lacunae that develop in the extraembryonic mesoderm. By this time, the primitive yolk sac is enveloped by chorionic cavity but for the portion where the embryo is attached to the trophoblast by the connecting stalk.

Gastrulation and Neurulation

The expansion of chorionic cavity reduces the size of primitive yolk sac, forming a *secondary yolk sac* above the hypoblast layer (Fig. 3.4). All the events till the formation of chorion and secondary yolk sac, occurs by the end of 2nd week. During the 3rd week of presomite period, *gastrulation* occurs. During gastrulation, the bilaminar disk is converted into trilaminar disk. The second and third week witness certain important changes like the formation of prochordal plate from the hypoblast layer by 2nd week and formation of primitive streak from the middle of epiblast layer during 3rd week. Primitive streak (Fig. 3.5) is a narrow trough with elevated margins, the cranial end of which develops into primitive pit surrounded by primitive node. The

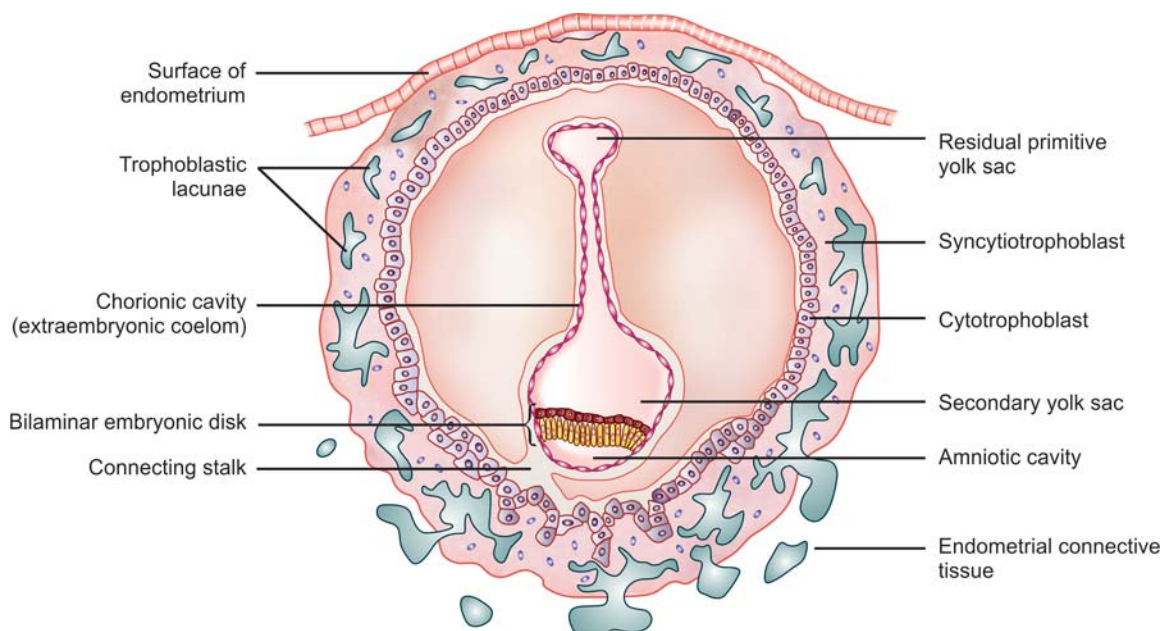


Fig. 3.4: Formation of secondary yolk sac in the bilaminar embryonic disk (13 days after fertilization)

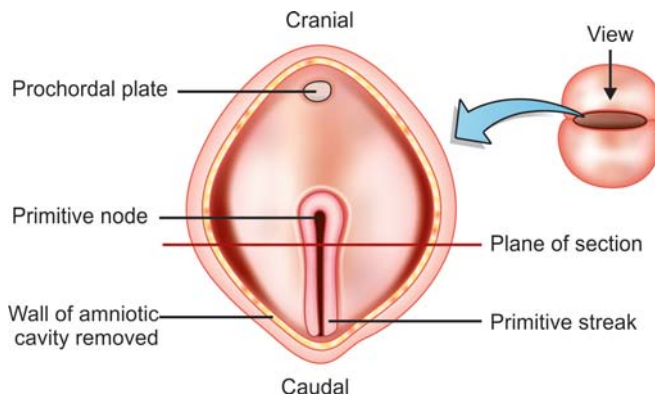


Fig. 3.5: Formation of primitive streak. Note the expansion of cranial end of the streak to form primitive node

prochordal plate is formed by 14th day of IUL. It is a thickening that later becomes the endodermal part of the oropharyngeal membrane. The formation of prochordal plate is achieved before the formation of the mesodermal layer.

The three germ layers, ectoderm, mesoderm and endoderm, are derivatives of epiblast layer. Cells of epiblast migrate and invaginate beneath the epiblast itself and form the endoderm. The outer layer or upper layer forms the ectoderm (Fig. 3.6). The layer of mesoderm is formed by the cells of epiblast invading between ectoderm and endoderm throughout the embryo except at the prochordal plate, future buccopharyngeal membrane, and the cloacal membrane. Cells of the primitive streak grow cranially to reach the prochordal plate to form the notochord which is a solid cylinder of cells, axial skeleton of the fetus forms around the notochord.

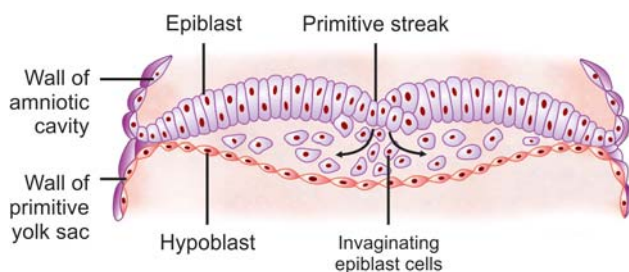


Fig. 3.6: Differentiation of three germ layers from the epiblastic layer. Invagination of epiblastic cells gives rise to endoderm

Neural Tube Formation

The process of development of the neural plate, neuroectoderm and folding to produce the neural tube is called as *neurulation*. The ectoderm above the notochord is induced to form a thickening called the neural plate. The midline of neural plate deepens to form a groove with elevated margins on either side, the neural folds. The neural folds grow towards each other and fuse to form the neural tube, which forms the central nervous system. The anterior end of the neural tube forms the fore, mid and hind brain. The edges of the neural fold on either side of neural groove are called *neural crests* (Fig. 3.7). Cells that proliferate from the neural crest undergo extensive migration between all the three germ layers and derivatives of neural crest cells are seen in the craniofacial region and the neck. Certain elevations develop in the area of hind brain, named rhombomeres. Neural crest cells migrate from specific rhombomeres to every specific location of the craniofacial region. Neural crest cells have their origin in the ectoderm but they have mesodermal properties.

Somite Period

Somite period is the period of organogenesis from 21st to 31st day of IUL. Anomalies in development would occur in this period. The visceral organs differentiate from mesoderm and endoderm. At this stage, there is rapid

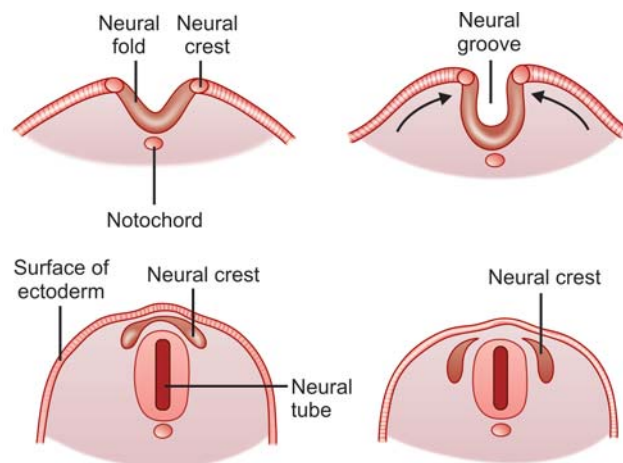
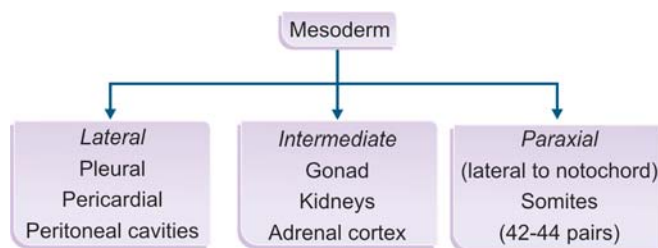
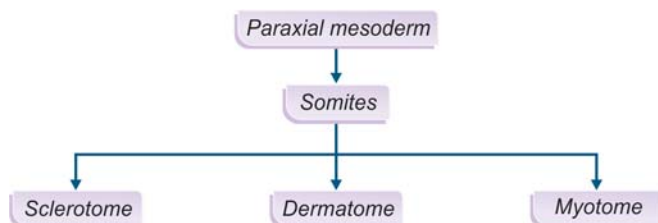
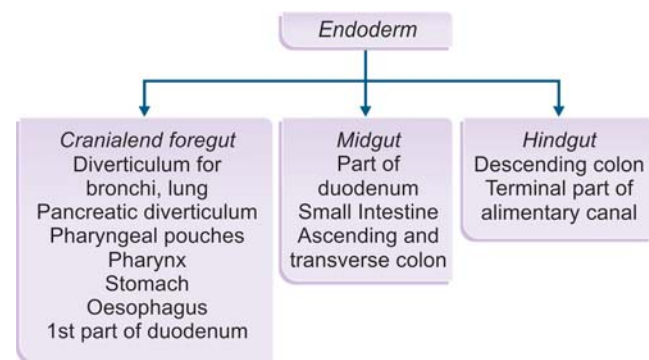


Fig. 3.7: Formation of neural tube (neurulation). The folding of ectoderm forms the neural fold, that deepens to form groove and then edges of the groove fuse to form the neural tube. The cells from the edge (crest) of the groove form the neural crest cells

Flow chart 3.2: Derivatives of mesoderm**Flow chart 3.3:** Derivatives of paraxial mesoderm**Flow chart 3.4:** Derivatives of endoderm

growth of the cranial end of the embryo, the caudal end lags behind. Thus the cephalocaudal gradient of growth is evident early in life. Head constitutes around $\frac{1}{2}$ of the total embryonic disk length at this stage. The end of presomite and the start of somite period witness the complete separation and establishment of all the three germ layers. The derivatives of mesoderm and endoderm are given in Flow charts 3.2 to 3.4.

Branchial Arches

Majority of the craniofacial skeleton is of ectomesenchymal origin whereas the other skeleton of the entire body is derived from mesoderm. In specific areas of the developing embryo, the migrating and

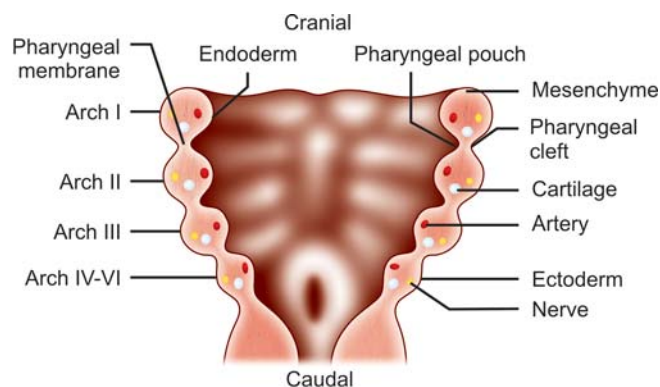


Fig. 3.8: Branchial arches. Pharyngeal pouch on the endodermal side and branchial groove on the ectodermal side are evident

rapidly proliferating ectomesenchymal cells develop elevations between ectoderm and endoderm. In the somite period, 4th week IUL, such elevations are seen in the ventral foregut resulting in the formation of six *pharyngeal arches* or *branchial arches* bilaterally, the fifth arch perishes; finally only five arches remain (Fig. 3.8). The arches are separated by 4 branchial grooves on the ectodermal aspect and 5 pharyngeal pouches on the endodermal aspect. Mesenchymal cells migrate between the ectoderm and endoderm and around the mesodermal condensation. Branchial arches decrease in size craniocaudally and when viewed from the frontal aspect they look like a stake of collars around the developing pharynx. Each branchial arch gives rise to some specific derivatives and every arch has the following structures: A cartilage rod, centrally located, forms the skeleton of the arch; a *branchiomere* that gives rise to the muscles of the arch; a vascular component, aortic arch artery, supplying the area; a nervous element, giving rise to sensory and specific visceral motor fibers for the cranial nerve supplying specific arch. The origin of the branchial arch components are given in Table 3.1.

Table 3.1: Branchial arch elements

S.No.	Branchial arch element	Derived from
1.	Cartilage	Neural crest derivative organized by pharyngeal endoderm
2.	Branchiomeric muscle component	Lateral plate mesoderm
3.	Muscles	Mesoderm core
4.	Blood vascular system	Lateral plate mesoderm

Table 3.2: Derivatives of branchial arches

S.No.	Arch	Skeleton	Muscle	Nerve	Blood vessel	Pharyngeal pouch
1.	First mandibular	Meckel's cartilage, maxilla, secondary palate, mandible, incus, malleus, anterior malleolar ligament, sphenomandibular ligament, spine of sphenoid	Muscles of mastication, mylohyoid, anterior belly of digastric, tensor tympani, tensor palatini	Mandibular division of trigeminal nerve (V cranial nerve)	Maxillary artery and part of external carotid artery	Tubotympanic recess forming auditory tube and middle ear cavity
2.	Second hyoid	Riechert's cartilage, styloid process, stapes, smaller (lesser horn) and superior part of body of sphenoid bone, stylohyoid ligament	Muscles of facial expression, stylohyoid, stapedius, posterior belly of digastric	Facial nerve (VII cranial nerve)	Stapedial artery probably later contributes to facial artery	Tonsillar fossa and palatine tonsil
3.	Third	Greater horn and lower part of body of hyoid bone	Stylopharyngeus	Glossopharyngeal nerve	Internal carotid artery	Inferior parathyroid, thymus
4.	Fourth	Thyroid cartilage, laryngeal cartilages	Constrictors of pharynx, cricothyroid, palatoglossus	Superior laryngeal nerve, pharyngeal plexus	Arch of aorta, right subclavian artery	Superior parathyroid
5.	Sixth	Laryngeal cartilages	Laryngeal muscles except cricothyroid	Inferior laryngeal nerve	Pulmonary artery	—

During the fetal period, due to rapid development of the fetus the branchial arch derivatives grow away from each other, still the nerve supply is retained hence the circuitous path of cranial nerves. The first branchial arch [mandibular arch] gives rise to two most important bones: maxilla and mandible. The second arch is called hyoid arch. The derivatives of branchial arches are given in Table 3.2.

Development of Face

In the region of the developing face, ectodermal thickenings called placodes arise at specific sites. Placodes later differentiate into special sense organs and form elements of the peripheral nervous system like ganglia, thus we have the optic/lens placode, otic placode, olfactory placode etc. At this time, the optic vesicle is located on the lateral aspect of the developing head. The migrating neural crest cells are divided into anterior and posterior streams when they confront the future eye. Anterior stream develops into a midventral elevation called the *frontonasal process* and the posterior stream develops into *branchial arches*. This stage of craniofacial

development presents the embryo with several elevations or processes, five such prominent processes bound a depression called stomodeum, the future site for the mouth. The processes are one centrally located frontonasal process, bilateral maxillary processes at the sides and bilateral mandibular processes below (Fig. 3.9). The maxillary and mandibular processes are derivatives of the first branchial arch. The first branchial arch is called the *mandibular arch*, the cartilaginous component is called *Meckel's cartilage* that forms in the somite period (41st-45th day of IUL) and acts as a scaffold for the development of mandible. Later, most of the cartilaginous substance disappears; mandible is largely ossified intramembranously except for mental ossicle, condyle and coronoid. Remnants of the cartilage are seen as ear ossicles—malleus and incus, spine of sphenoid, two ligaments namely anterior ligament of malleus and sphenomandibular ligament.

The cranially located frontonasal process forms the forehead and nose; maxillary process forms the maxilla, lateral part of the face, zygomatic bones, etc. and mandibular process gives rise to mandible. At this

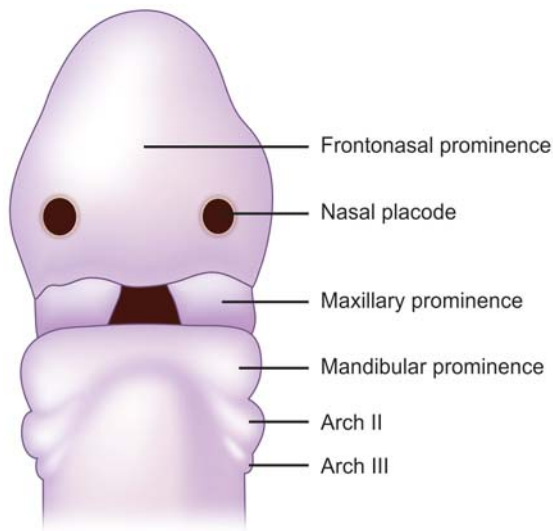


Fig. 3.9: Developing face. Frontonasal process in the cranial end with nasal placodes, maxillary process on either side and mandibular process below are seen bounding the stomodeum

junction, the stomodeum that is bound by the five processes is wide, so is the frontonasal process. At the inferior aspect of the frontonasal process, bilaterally there is development of two *placodes* (ectodermal thickenings) called nasal/*olfactory placodes*. The frontonasal process develops two elevations on either side of the placodes called the medial and lateral nasal processes (Fig. 3.10). The rapid development of these two processes sinks the nasal placode to form the nasal pit that is the future anterior nares. By 5th week, the nasal pit which is initially separated from the oral cavity by the oronasal membrane

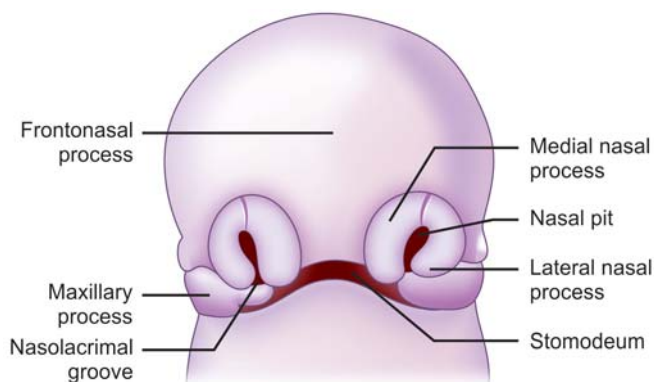


Fig. 3.10: Developing face. Medial and lateral nasal processes around the nasal pit, maxillary process growing medially to meet the MNP

opens into the primitive oral cavity by disintegration of the same membrane. The stomodeum establishes continuity with the pharyngeal cavity by the rupture of *buccopharyngeal membrane* [bilaminar] at 27th day of IUL. The two medial nasal processes grow towards each other and fuse at the midline; from now on it is called *globular process*. The derivatives of globular process are tip of the nose, columella, philtrum, prolabium, primary palate (with 4 maxillary incisors).

The maxillary process grows ventromedially to fuse with the medial nasal process and forms the rest of the upper lip (Fig. 3.11). The fusion is not immediate; there is an initial epithelial fusion into a single layer, a common epithelial wall called the nasal fin. It disintegrates eventually due to the migration of mesenchyme. The maxillary mesenchyme merges with the mesenchyme of the globular process. The maxillary process joins also with the lateral nasal process, the junction being marked by the naso-optic furrow. The furrow develops into a canal called nasolacrimal duct connecting the conjunctival sac to the lateral wall of the nose. The lateral nasal process provides for the alar portion of the nose. The maxillary process contributes to the lateral aspect of the upper lip, cheek, maxilla, rest of the maxillary teeth and secondary palate. The nasal septum develops in the midline as a projection from the cranial base cartilage in the forebrain region. The fusion of medial nasal processes into the midline structure called globular process narrows the frontonasal process, at the same time lateral aspect of the face is overgrowing, resulting in the redirection of the eye or optic placode from lateral to frontal direction. Thus the stomodeum is narrowed

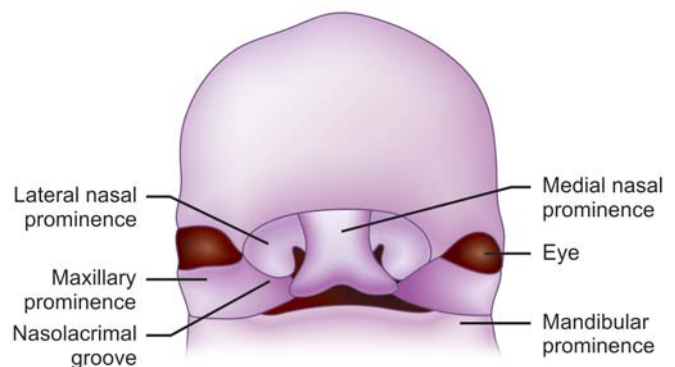


Fig. 3.11: Median nasal process of both sides merges to form a median globular process. Stomodeum has narrowed

further. In the mean time, stomodeum becomes continuous with the gut by the disintegration of buccopharyngeal membrane at about 27th day of IUL. The oral, nasal and pharyngeal cavities are a single chamber with developing cartilaginous cranial base as the roof. The mandibular processes grow towards each other and fuse in the midline.

Postsomite Period

In the postsomite period, the organs and systems formed during the somite period increase in size and the external body form is established. The period extends from 4th-8th week IUL. In the craniofacial region, the cranial vault and base are delineated and the basic maxillary and mandibular bony forms are established, tongue develops, palatal fusion occurs.

The fusion of maxillary process with medial nasal process and the two mandibular processes narrows the stomodeum. Mouth is continuous now with the developing oral cavity due to the rupture of buccopharyngeal membrane. Lateral palatal shelves develop as outgrowths from the internal aspect of maxillary processes. Palatal shelves form the future secondary palate. At this stage, the oral cavity is comparatively small; the volume of oral cavity is occupied by the developing tongue.

Development of Tongue

The tongue is derived from the first four branchial arches. Tongue develops from the ventral wall of oropharynx, in the form of two lingual swellings and tuberculum impar behind the lingual swellings. Anterior 2/3rd of the tongue is formed from the lingual swellings derived from the 1st arch. Endodermal prominence from 2nd, 3rd, and 4th arches form a midventral copula. The posterior end of copula is called hypobranchial eminence which ultimately forms the posterior 1/3rd of the tongue. The junction of anterior and posterior parts is marked by sulcus terminalis, the central part of the sulcus bears the foramen caecum from which thyroid diverticulum originates. During early stage of development of tongue, the lingual swellings enlarge and fuse with each other overgrowing the tuberculum impar which sinks behind (Fig. 3.12). The lingual swellings provide for the ectodermally derived mucosa of the anterior 2/3rd and hypobranchial eminence forms the endodermal lining

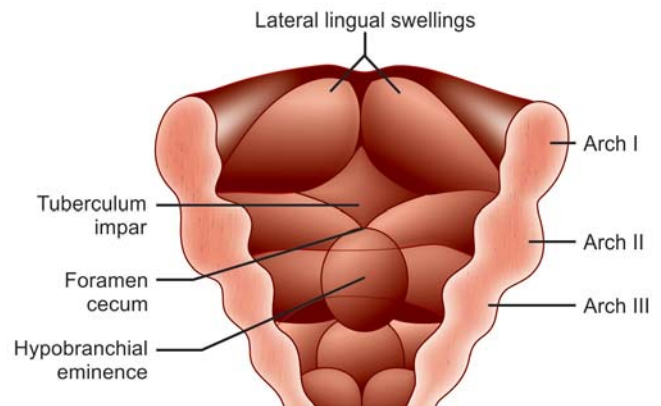


Fig. 3.12: Development of tongue. Between the branchial arches in the floor of the primitive oral cavity, lingual swellings, tuberculum impar develop. Hypobranchial eminence forms the posterior part of the tongue

of posterior 1/3rd. The nerve supply varies thus for the anterior and posterior parts. The muscles of the tongue are derived from occipital somites. Occipital somites are located in the floor of the pharynx dorsally. As the muscle mass of the future tongue migrate forward towards the developing mucosal sac of the tongue, they carry the hypoglossal nerve along. Papillae of the tongue start developing at 2nd month of IUL. First to form are the fungiform papillae at around 11 weeks of IUL over the dorsum of the tongue. Circumvallate papillae develop in front of the sulcus terminalis at 2nd-5th month IUL. Filiform papillae are matured only postnatally. Taste buds, epithelial in origin, start developing by invasion of epithelium by nerve cells at around 7th week IUL. They mature by 13-18 week *in utero*, thus taste is initiated in the intra uterine life. At the time of birth all the fungiform papillae have taste buds. The nerve supply of tongue is summarized in Table 3.3. At this stage, tongue encroaches the primitive oral cavity from the floor to roof because the volume of oral cavity is comparatively small. At 6 weeks of IUL, the developing lateral palatal shelves from the inner margin of maxillary process are hampered by the large tongue from growing horizontally; instead they grow down vertically on either side of the tongue (Fig. 3.13). The discrepancy between the size of tongue and oral cavity is still persistent at birth; the infant's tongue is relatively large and protrudes out to help in suckling.

Table 3.3: Nerve supply of tongue

S. No.	Part of tongue	Nerve supply
1.	Anterior 2/3rd sensory gustatory	Lingual nerve (1st arch) Chorda tympani (2nd arch)
2.	Posterior 1/3rd sensory gustatory	Both by glossopharyngeal nerve (3rd arch)
3.	Muscles of tongue (extrinsic and intrinsic except palatoglossus)	Hypoglossal nerve (occipital somites) except palatoglossus
4.	Palatoglossus	Pharyngeal plexus-glossopharyngeal nerve

Secondary Palate Development

During 7-8th week of IUL, descent of the tongue leads to elevation of the lateral palatal shelves. Numerous hypotheses have been put forth to explain the phenomenon of palatal elevation, that precedes by a week in boys. The elevation of vertical palatal shelves to a horizontal position starts around 7th week of IUL and the phenomenon has been ascribed to withdrawal of developing face from the heart prominence. Head is bent over the heart prominence; elevation of the face facilitates growth of mandible thus increasing the volume of oral cavity. Tongue senses the increase in space and descends down leading to elevation of palatal shelves (Fig. 3.14).

The elevation of palatal shelves may also be due to the change in the biochemistry of oral cavity; change in physical consistency of connective tissue; variation in vasculature and blood flow; rapid differential mitotic growth; intrinsic shelf force; change in pressure between nasal and oral region due to tongue contraction and movements.

After palatal elevation, the lateral palatal shelves approximate with member of the opposite side, the nasal septum above and primary palate (ingrowth of frontonasal process) in front. The palatal shelves swing from vertical to horizontal position. The fusion of palatal shelves does not start from the anterior terminus rather it begins about 1/3rd the distance from anterior margin at the future site of incisive papilla. Fusion proceeds both anteriorly and posteriorly from that region. Fusion starts at 8th week and is complete by about 12th week of IUL. Nasal septum fuses with palate only anteriorly, in

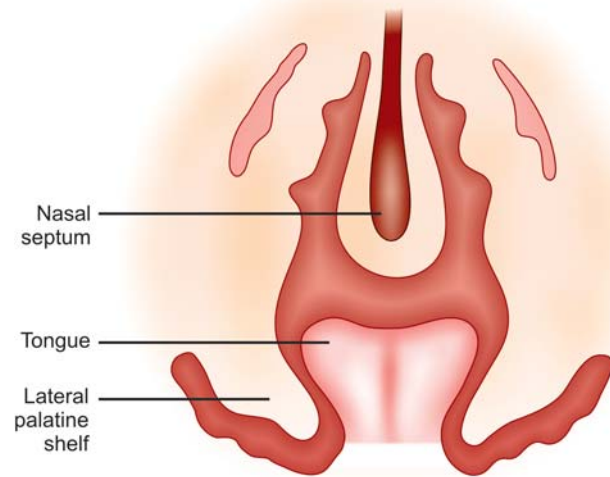


Fig. 3.13: Developing secondary palate. The palatal shelves are vertically oriented with the intervening tongue that extends till the roof of oral cavity

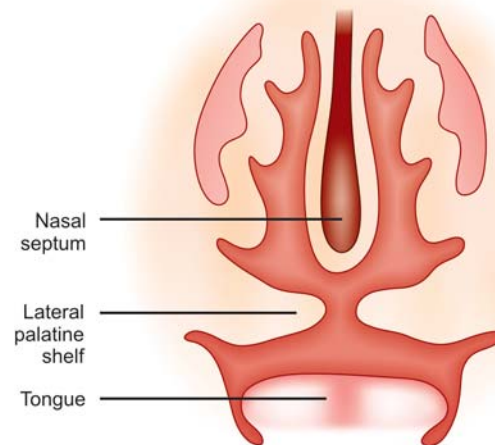
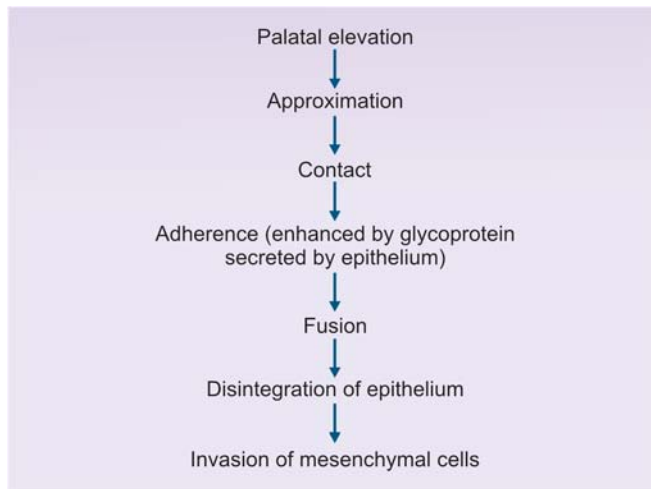


Fig. 3.14: Elevation of palate. Descent of the tongue leads to elevation of palatal shelves

the posterior region, the soft palate and uvula remains free. Fusion at first is only epithelial, the epithelial layers are thickened and they approximate and fuse to form a single layer of epithelium. The sequences of events are given in Flow chart 3.5.

The disintegration or degeneration of epithelium is a highly ordered process. It is actually programmed cell death or apoptosis. The site of fusion is the future

Flow chart 3.5: Palatal elevation: Sequence of events

midpalatal suture. Ossification starts by 8th week of IUL. There is only one center of ossification for each maxilla. Posterior part of the palate receives ossification center from the palatine bone. Posterior most part remains unossified as soft palate and uvula. Initially, the palatal arch is very shallow, the depth is gained postnatally. From 7-18 weeks, palate increases in length. At 4th month, palate grows more in width along the midpalatal suture than length so that the width and length at birth are almost equal. Deepening of palatal vault occurs postnatally by apposition at the alveolar margin and the palatal vault.

Development of Mandible

Mandible, derived from the 1st arch, arises as bilateral processes that grow ventromedially and fuse in the midline. Mandibular process together with the maxillary and frontonasal processes bound the primitive mouth/stomodeum. At about 5th week IUL, bilateral rod like cartilaginous condensations develop from the site of future ear to the midline but this does not imply that mandible ossifies endochondrally. Actually, mesenchymal condensations develop lateral to *Meckel's cartilage* at 36-38 days of IUL and ossification starts from the same location near the future mental foramen at 6th week of IUL. As the ossification proceeds medially towards the midline, Meckel's cartilage largely disappears and what is left of it becomes the ear ossicles—malleus and incus, spine of sphenoid, anterior ligament of malleus,

sphenomandibular ligament and genial tubercle of the symphyseal region. The first structure to form before mesenchymal condensation is the inferior alveolar nerve. The *neurotrophic* influence seems essential for mesenchymal condensation and initiation of ossification. Ossification starts lateral to the region where inferior alveolar nerve bifurcates into two. Though ossification of mandible occurs intramembranously, some part of it is formed by endochondral ossification. Cartilages of coronoid process and mental ossicles develop around 14th week of IUL. Coronoid and condylar cartilages grow in size, coronoid cartilage ossifies and fuses with the ramal portion of mandible; cartilage in the mental region also ossifies, the cartilage becomes incorporated in the symphyseal region.

At 10th week of IUL (fetal period), condylar cartilaginous condensation appears as cone shaped projection. The cartilage cells of the condylar process differentiate and increase in number, thus increasing the size of the condyle. Cartilage undergoes both interstitial and appositional growth. Ossification starts at 14th week of IUL. Most of the cartilaginous substance is replaced with bone by about the midfetal period. The superior end of condyle persists as cartilage. The presence of cartilage in the superior end may be responsible for the growth of mandible in the postnatal life.

The left and right sides of mandible are separate at birth. The ramus of mandible is relatively short and low. The coronoid and condylar process of the fetal mandible are at the level of the occlusal plane. Coronoid process is bigger than the condylar process. In the postnatal life, height is gained by alveolar and ramal growth. The mandibular canal is low in position. The buds of deciduous teeth are contained in the mandibular corpus.

Neurocranium/Calvarium

The cranial vault and cranial base start developing in the early stage of embryo but ossification of bones start in the postsomite period and extend well into the period of fetus. In fact, ossification of most of the bones complete only postnatally. To avoid confusion the development of both are thus explained in the postsomite period.

In the somite period, around the developing neural tube (future brain), mesenchymal condensations appear to form a two layered capsule, the outer called the ectomeninx and the inner capsule forms the

endomeninx. Endomeninx forms the pia and arachnoid membrane around the brain while ectomeninx forms the duramater that surrounds the brain and the calvarial bones and the bones of cranial base. All the calvarial bones ossify intramembranously while the cranial base ossifies by endochondral ossification. The dura is strongly attached to the bones of the vault by means of fibers in the areas of sutures. The rapidly growing brain serves as a functional matrix for the expansion of the ectomeninx. Ossification centers for the cranial bones develop in the outer layer of the ectomeninx. Almost all the bones start ossification around 8th week of IUL. Frontal bone develops as two separate bones with ossification center appearing in the superciliary arch region, one on either side at 8th week of IUL. Secondary centers appear for the frontal bone, all of which fuse by 6-7 months of IUL. Parietal bone develops ossification center one on either side near the eminence. Squamous temporal bone ossifies intramembranously with one center at the zygomatic process, tympanic ring also ossifies intramembranously, and rest of the temporal ossifies endochondrally. Similarly, the squamous portion of occipital bone undergoes intramembranous ossification with one center appearing in the 8th week of IUL.

At birth, ossification is not complete; all the cranial bones are separated by fibrous tissue. The sutures are not mature and the corners around the parietal bone and its junction with other bones is not ossified but covered with fibrous tissue, such areas are called fontanelles.

Cranial Base

The development of chondrocranium or the cranial base is the most complex of all the craniofacial structures. The adult cranial base is like a perforated plate with number of foramina for the exit and entry of cranial nerves and vessels. Cranial base ossifies by endochondral ossification. The cranial base consists of occipital bone at the posterior end, undersurface of body and greater wing of sphenoid, undersurface of petrous temporal. Development of cranial base commences at 4th week of IUL with mesenchymal condensation between the foregut and the developing brain [neural tube]. The mesenchymal condensation of the outer layer of ectomeninx chondrifies at 40th day of IUL.

Cephalic flexure begins at about 5th week of IUL due to the rapid growth of neural tube and division of

prosencephalon into telencephalon and diencephalons. The flexure of brain bends the notochord as well. Cartilages form initially on either side of the notochord. These are called parachordal cartilages, extending from hypophysis to caudal end of hindbrain. Immediately posterior to the parachordal cartilages on either side of rhombencephalon, there is formation of sclerotomes from the occipital somites. The sclerotomes bound the foramen magnum, contributing to the occipital condyles. Pontine flexure [6th week of IUL] paves way for the division of rhombencephalon into metencephalon and myelencephalon, in the process flattening the notochord; parachordal cartilages merge with occipital sclerotomes.

Hypophyseal pouch (Rathke's pouch) arises as an ectodermal invagination from the stomodeum/roof of the primitive oral cavity; it forms the anterior lobe of pituitary gland. It is at the cranial end of the notochord. Immediately behind the hypophyseal pouch are postsphenoid (polar) cartilages. Polar cartilages give rise to posterior part of body of sphenoid and sella turcica.

Two presphenoid [trabecular] cartilages develop from prechordal condensations, they fuse to form the anterior part of body of sphenoid bone. Anteriorly, the presphenoid cartilages contribute to the formation of mesethmoid cartilages, which in turn forms the perpendicular plate of ethmoid and crista galli. Lateral to hypophyseal pouch are the orbitosphenoid and alisphenoid cartilages, they form the lesser and greater wings of sphenoid respectively. Nasal capsule chondrifies around 2nd month of IUL. Nasal capsule expands into a box-shaped structure that is cartilaginous. The nasal septum is cartilaginous but for the posteroinferior part that forms a membranous vomer. The cartilage of nasal septum remains patent and contributes to the anterior and inferior growth of midface. In the lateral wall of the nasal capsule, there is development of the ethmoidal and inferior nasal conchae by cartilaginous ossification. The otic capsule undergoes chondrification to form the mastoid and petrous portion of temporal bone. Basisphenoid starts ossification at 10th week of IUL. All the cartilages constitute the basal plate, which is an irregular mass of cartilage; the nerves exiting and arteries entering the cranial base are established before ossification commences and hence the basal plate is highly perforated. Ossification centers are numerous for the cranial base bones. An insight into the number of ossification centers, chronology of ossification and

Table 3.4: Ossification of cranial bones

S. No.	Bone	Parts/No. of centers	Chronology of ossification
1.	Occipital center: 7	Squamous: 4 Basilar: 1 Condylar: 2	Supranuchal: Intramem 8th week IUL Infranuchal: endo 10th week IUL Endo: 11th week IUL Endo: 12th week IUL
2.	Temporal (bilateral) center: 11	Squamous: 1 Tympanic: 4 Petromastoid: 4 Styloid: 2 Mastoid postnatal	Intramem: 8th week IUL Intramem: 3rd month IUL Endo: 5th month IUL Endo: Before and after birth
3.	Ethmoid center: 3	Median: 1 Mesethmoid Lateral: 2 Nasal capsule	Endo: Before birth Endo: 4th month IUL
4.	Inferior nasal concha center: 1	Nasal capsule: 1	Endo: 5th month IUL
5.	Sphenoid center: 15	Greater wing and lateral pterygoid plate: 2 Presphenoid: 5 (1 median and 2 paired) Postsphenoid: 4 (2 IN each hypophyseal cartilage) Medial pterygoid: 2 Synchondrosis	Intramem: 8th week IUL Endo: 4th month IUL Endo: 4th month IUL Fuses before birth
6.	Frontal center: 2	at superciliary arch on each side: 2	Intramem: 8th week IUL
7.	Parietal (bilateral) center: 1	At parietal prominence: 1	Intramem: 8th week IUL

method of ossification of all the cranial bones is given in Table 3.4.

FETAL PERIOD

Fetal period is from the 3rd month IUL till birth. Fetal of period is marked by rapid increase in the size of the fetus.

DEVELOPMENT OF TEMPOROMANDIBULAR JOINT (TMJ)

Temporomandibular joint starts its development as two separate surfaces; condylar and temporal. The primary joint for jaw movement in the embryonic period is the joint between malleus and incus but as the development of ear proceeds, malleus and incus lose contact with the Meckel's cartilage. Condylar cartilage develops at 10th week of IUL. The cartilaginous proliferation leads to growth of condylar projection towards the temporal

bone. The mesenchyme between the two bones differentiates into fibrous tissue. By 12th week of IUL, two joint cavities are delineated by intervening fibrous articular disk. After cavitation, synovial membrane invades and lines the joint cavities, it secretes the synovial fluid. Progressive development of the joint occurs and the articular disk by its compression in the center leads to a biconcave shape.

At birth, mandibular fossa is flat and articular eminence is not developed. This configuration helps in anteroposterior movement of the mandible during suckling. Articular eminence starts development with the eruption of deciduous teeth.

CHANGING RELATIONSHIPS IN FETAL FACE

Growth of the fetal craniofacial skeleton is isometric at places and allometric at others. Isometric growth is proportionate increase in size so that the relation of the

parts with each other remains the same. Allometric growth on the other hand is the change in shape and proportions that changes relationship between parts. Isometric growth is seen in calvarium, anterior cranial base etc. posterior cranial base does not grow as well as the rest of the cranium in the prenatal period but postnatally it grows by growth at synchondrosis. Allometric growth is evident in the prenatal maxillomandibular relation in all the three planes: sagittal, transverse and vertical. Anteroposterior relation undergoes extensive alteration between embryonic and fetal periods. Diewert (AJO 1985) published the result of his studies on a large collection of staged human embryos and fetuses. The maxillomandibular relation was assessed by the angle formed by lines from the maxillary and mandibular incisor bud to nasion. Point A and B are not evident in fetal skeleton hence the tooth buds are taken to assess the sagittal relation. The study revealed that before secondary palate formation, the MxI-N-MdI angle was large and positive, 10° . In the early stages, embryonic form is bent like the letter C, hence anterior cranial base and mandible are retrusive. With the elevation of face from heart prominence and thoracic wall, there is spurt of growth in maxillomandibular region. Descent of tongue is partly due to mandibular growth that is so great that the angle MxI-N-MdI becomes negative -3° . Thus this angle is an analogue of ANB in prenatal period. The greatest prominence of maxilla is seen in early fetal period when maxillary angle to cranial base is $85-87^\circ$, a 25° increase from the early embryonic period. During this period, maxillomandibular relation is 10° but eventually decreases to -3° partly due to decline in maxillary growth and catch up of mandibular growth.

There is decline in mandibular growth after palatal elevation; maxillomandibular angle of 8° is regained at 12 weeks of IUL which is maintained thereafter. The spurt of mandibular growth is attributed to the growth of Meckel's cartilage mouth opening reflexes starting in embryonic period, accompanying muscular contraction leads to descent of tongue. The newly established maxillary prominence with change in profile is maintained in the second and third trimester.

There is prominent sagittal growth in the first trimester; it is compensated by an increase in width in the 2nd and 3rd trimester. The fetal face broadens. Sagittal growth of mandible is so minimal that sagittal angle increases to 15° , there is only isometric growth from then onwards.

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Principles of Growth

CHAPTER OUTLINE

- **Growth Pattern**
 - Cephalocaudal growth
 - Scammon's growth gradient
 - Variability
 - Concept of normality and variability
 - Timing, distance and velocity curves
- **Mechanism of Bone Growth**
 - Deposition and resorption
 - Endosteal and periosteal bone growth
 - Remodeling
- **Growth Movements**
 - Drift and displacement
 - V principle
 - Surface principle
 - Posterior growth and anterior displacement
- **Growth Equivalents Concept/Enlow Counterpart Principle**

Craniofacial growth is a complex process. A thorough understanding of the principles or concepts of growth will enable the dentist, orthodontist, pediatric dentist and oral surgeon to meticulously plan the treatment, and also to understand the normal variations from abnormalities. *Growth* is usually defined as the increase in size (Todd) but tend to be linked more to change than anything else. According to Moyers, growth may be defined as the normal changes in amount of living substance. Growth is the result of biologic processes by means of which living matter normally gets larger. Other definitions of growth are as follows. Krogman defines growth as increase in size, change in facial proportions over time. Moss: defines growth as any change in morphology which is within measurable parameter.

Growth is quantitative, i.e. it is a measurable aspect of biologic life. The units of growth are inches per year

or grams per day. Characteristically growth is equated with enlargement. But sometimes there are instances in which there is decrease in size during growth, e.g. thymus gland shrinks after puberty. Growth highlights the normal dimensional changes over period of time. Growth might cause change in form or proportion, increase or decrease in size, change in texture, complexity. In simple words, growth is change or difference in quantity.

Development denotes an increasing degree of organization, often related to consequences for the natural environment. Development refers to the naturally occurring unidirectional changes in life of an individual from its existence as a single cell to its elaboration as a multifunctional unit terminating in death. It encompasses the normal sequential events between fertilization and death. The life of an individual starts from the primordial germ cells giving rise to gametes. The term 'Multifactorial Unit' emphasizes the functions rather than the multiple cellularity. Moss states that "Development can be considered as a continuum of causally related events from the fertilization of ovum onwards". Development includes all the changes in life of a subject from his origin as a single cell till death. It comprises sequential events from fertilization till death. Development = Growth + Differentiation + Translocation, where differentiation means change in quality, and translocation means change in position.

Profit differentiates the term growth and development as follows. The basic difference between growth and development is growth can be considered an "anatomic phenomenon" whereas development is a "physiological and behavioral phenomenon".

Meredith on the contrary, defines growth as entire series of anatomic and physiologic changes taking place

between the beginning of prenatal life and close of senility. He does not consider growth and development as separate entities.

Differentiation is the change from generalized cells or tissues to more specialized kinds during development. Differentiation is change in quality or kind. *Translocation* is change in position. Translocation of chin point downward and forward is far more than any growth at the chin itself. *Maturation* is the qualitative changes which occur with ripening or aging.

GROWTH PATTERN

Pattern refers to the way in which the various parts of the body are arranged in a proportional relationship. It represents the set of proportional relationships and not a single proportional relationship. The relationships are not only represented at a particular point of time but also portray the change in relationship over time. Pattern includes arrangement of parts, value, or events; arbitrary lists of statistics; or relations among measurements. This term explains the persistence or invariance in contrast to the word growth, which implies increase in size. Moyers defines pattern as a set of

constraints operating to preserve the integration of parts under varying conditions or through time.

Cephalocaudal Growth

Cephalocaudal growth (Fig. 4.1) gradient is an example of change in the body proportions that occurs in normal growth and development.

- In the third month of intrauterine life, head constitutes 50 percent of the total body length. The cranium is large, relative to the face and represents more than half of the total head. Limbs are underdeveloped. At the time of birth the trunk and the limbs have grown faster than head and face, so that the portion of the head is decreased to 30 percent. In the adult, there is progressive reduction in relative size of head which is 12 percent of the total head body length. Thus there is always an increase in the gradient of growth towards the caudal direction right from the intrauterine life. We could also figure out that greater proportion of head which is seen during birth is reduced in the adult. Even in the head and face at the time of birth, there is a larger cranium and a much smaller face. This increased axis of growth in

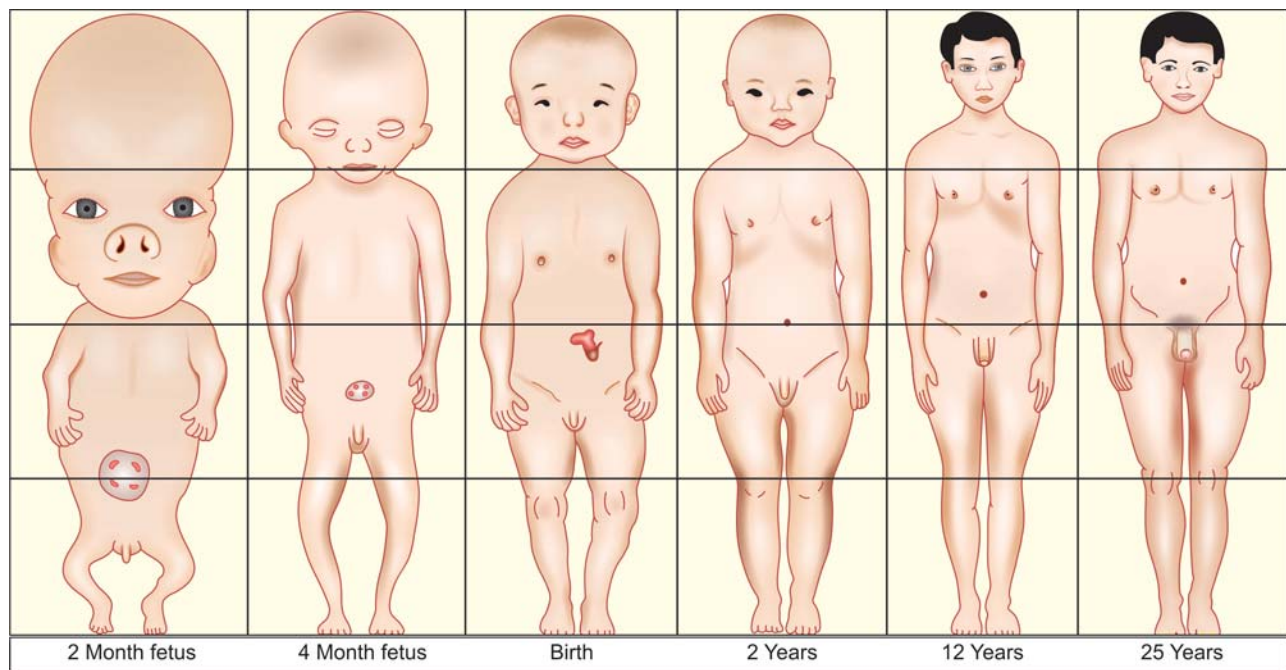


Fig. 4.1: Diagrammatic representation of cephalocaudal growth. Note the proportional increase in growth of the extremities as age advances

the caudal direction is called as cephalocaudal growth gradient. Cephalocaudal growth is evident in the face also. At birth, jaws and face are less developed when compared to skull, maxilla being closer to head, grows faster and growth is completed before mandibular growth. Mandible being away from the brain grows more and growth completes later than maxilla.

Scammon's Growth Gradient (Fig. 4.2)

Human body is comprised of four major tissues. They are neural, somatic—includes muscles and bone, lymphoid and genital/sexual tissue. Not all the tissue systems of the body grow at the same rate. Growth of the neural tissues is complete by 6 or 7 years of age. General body tissues, including muscle bone and viscera show an 'S' shaped curve, with a definite slowing down of the rate of growth during childhood and acceleration at puberty. Lymphoid tissues proliferate far beyond the adult amount in late childhood and then undergo involution at the same time when growth of the genital tissues accelerates rapidly.

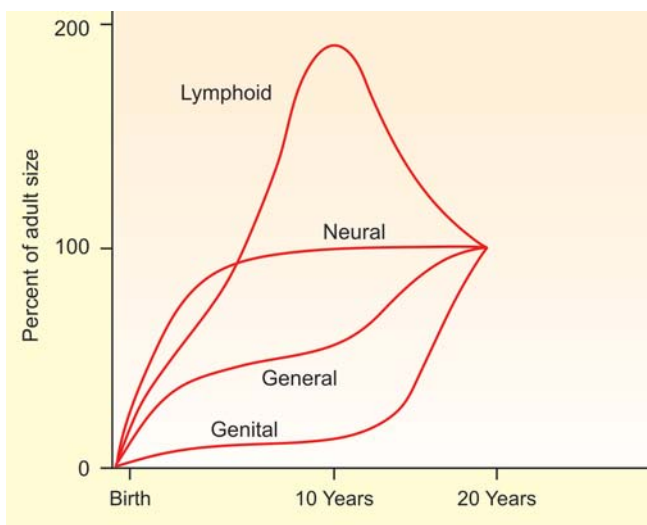


Fig. 4.2: Scammon's curves for the different tissue systems in the body. *Neural tissue* growth completes by 6-7 years of age. *General body tissues* follow a 'S' shaped pattern. Slowing of growth during childhood and acceleration at puberty is seen. *Lymphoid:* Attains peak growth and grows beyond adult amount during late childhood and undergoes regression at puberty or when genital growth acceleration takes place. *Genital:* Secondary sexual characteristics begin to appear during puberty and reaches peak by 20 years of age

Variability

Another important concept of growth is variability. According to Moyers, variability is the law of nature. No two individuals grow in the same manner. It is very difficult to say whether an individual growth is within the normal range, or at the extremes of normal range or out of normal range. Variations can be attributed to both genetic and environmental factors. Variations in growth can be expressed by statistics as range of differences found in a population containing people of similar age, sex, socioeconomic background and race. Before understanding the concept of variability it is necessary to understand what normalcy stands for.

Concept of Normality and Variability

Normal refers to that which is usually expected, is ordinarily seen, or is typical. The usage of the word normal and the concept normality varies and is often a source of misunderstanding. Normality can be explained by the following ways.

- **Statistics:** These are specific mathematical ways for portraying the central tendency of a group or population. The parameters mean, median and mode and the standard deviation are used to express the normality.
- **Evolutionary:** Since all forms of life have crossed the critical test of survival, abnormal forms that are unable to cope have been lost.
- **Functional:** It is normal for every organism to establish effective homeostasis with the environment in order to adapt and survive.
- **Esthetic:** Facial characteristics appearing esthetic to one type of culture may not be applicable to another one. The feet of baby girls are warped, distorted growth, insertion of wooden plate in lips, scarring of the face has been practiced in different cultural groups.
- **Clinical:** There are certain axioms which are needed to be followed when explaining normality in a clinical perspective. Normality must not be equated with ideal or the desired, nor is it appropriate as a goal of treatment of an individual.

It is more appropriate to put forth **variability** as deviation from the usual pattern, and express it in a quantitative manner. This can be done with the help of a growth chart, where a child is evaluated in relation

to their peers on a standard growth chart. The growth charts commonly used are for height and weight for age, length for age, head circumference for age, BMI for age, and weight for stature. The chart has solid lines on the graph which depicts how far an individual varies from the usual pattern. An individual who is exactly at the midpoint of the normal distribution would fall along the 50 percent line of the graph. One who is larger than 90 percent of the population would plot above the 90 percent line; one who is smaller than 90 percent of the population would plot below the 10 percent line.

The variability from the charts can be predicted in two ways. First, the location of the individual in the graph relative to the group can be established. Child who is beyond 97 percent of the population should receive special study before being accepted as just an extreme of the normal population.

Second, the charts can be used to follow a child over time to evaluate whether there is an unexpected change in growth pattern. Since the pattern is predictable, the child's growth should plot along the same percentile position of individual, relative to his or her peer group changes. If there is a marked change, an abnormality should be suspected. Variability in growth is usually due to the following reasons:

- Variations within normal range.
- Variations due to other influences, e.g. illness, malnutrition.
- Variations due to timing effects.

Timing

Another major concept in physical growth and development is timing. The periods during which the growth processes are turned on are called growth spurts, of which the pubertal growth spurt is much important. Variability in timing is seen usually in relation to sex, age, body build, in relation to cultures, etc. Some children grow and mature early when their contemporaries are beginning to catch up. Others grow slowly, but catch up later and sometimes even surpass the peers who were once larger. Children undergo a spurt in growth at adolescence; this can be demonstrated by using growth curves, which uses height or weight against age, or amount of increase in height per year against age (velocity curve).

Velocity and Distance Curves

These are obtained by measuring individual children repeatedly during their growth. Height and weight are the most commonly used measurements. The other measurements used are head circumference, sitting height and skinfold thickness. The curve obtained by joining successive points of a child's height from birth to adulthood is called *distance curve*. A standard distance curve will show rapid growth upto 2 years, then a steady increase in height upto 13 years, thereafter an acceleration due to pubertal spurt for a brief period and then a slow down until adulthood is achieved. The earliest study about adolescent growth spurt started in 1759 when Gueneau de Montbeillard began a 18 year study on his son to determine the yearly increments of growth. He plotted the height of his son against age in years (Fig. 4.3). This is a distance curve where the height was plotted against age. In the distance curve, the height measured at set intervals was plotted against time. The resultant graph helps us to visualize the entire pattern of growth from the first to the last. Since the increase in height stops after the adolescent growth spurt, the distance curve finally ends in a plateau.

Growth variability, because of timing can be seen particularly in girls, who mature sexually earlier than boys. This can be best explained by velocity curves. In



Fig. 4.3: A point-to-point curve of the 18th century data of Montbeillard on the growth in height of a single individual

velocity curves, amount of increment in height per year is plotted against age in years. The velocity curve, rather than a distance curve gives more information about the acceleration and deceleration in the growth rate. Velocity curves are more revealing than the distance curves. It is unlikely to be possible to measure a child at exact intervals of a year and, so it is best to convert ages in days and months into decimals. The growth achieved between certain dates can then be divided by the decimal figure obtained by subtracting the first date from the last, giving the velocity in mm/year (Fig. 4.4).

Variability in timing also affects age. Age is measured chronologically since the time of birth or conception. Not all individual in the same chronological age have the same maturational status. To remove this variability, developmental age can be used. It is possible to measure age biologically towards various developmental markers or stages. In a velocity curve, if we substitute stages of sexual development for chronological age, we could find out that early average or late maturers all follow a same pattern. This would not be possible if chronological age was used.

MECHANISM OF BONE GROWTH

Bone growth is based on certain basic principles. Bones do not enlarge symmetrically, but grow by complex differential mechanisms. Two mechanisms are important for bone growth. Direct bone growth by means of deposition and resorption processes on the bone

surfaces, which cause the cortical plate to drift. Displacement of the entire bone occurs due to growth of the bone itself or expansion of adjacent structures.

Deposition and Resorption

Bones grow by addition of new bone tissue on one side of the bony cortex and taking it away from the other side (Fig. 4.5). The surface facing towards the direction of progressive growth receives new bone deposition. The surface facing away undergoes resorption. The outside and the inside surfaces of a bone are covered by irregular patterns called growth fields. It is comprised of various soft tissue osteogenic membranes or cartilages. Bone does not grow by itself. Bone growth is influenced by this soft tissue growth fields. The genetic program of the bone growth is not contained within the hard tissue. But it resides in the surrounding tissue growth fields. All bones have got both resorptive and depository fields.

The varying activity of the depository fields is responsible for the differential growth processes, resulting in bones of irregular shapes. The irregularity is the result of variety of functions imposed on the bone by attachments, sutural articulations with other bones, insertion of teeth, and other processes. All the resorptive and depository growth fields throughout a bone do not have the same rate of growth activity. Some depository fields grow more rapidly or to a much greater extent than others. Same is true for resorptive fields. Fields that have some special significance or noteworthy role in the

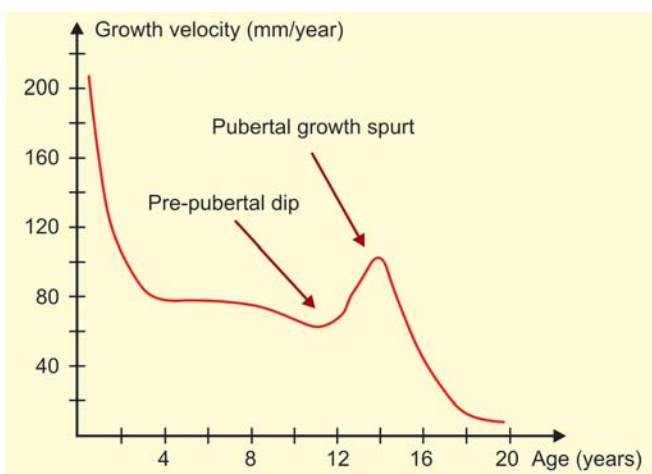


Fig. 4.4: Standard velocity curve for a child with normal growth

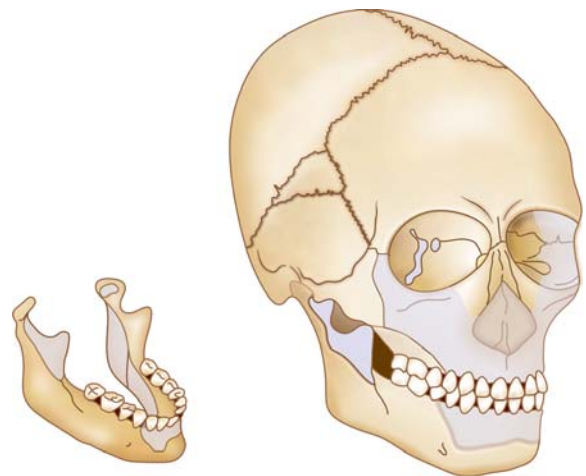


Fig. 4.5: Sites of deposition and resorption. Brown fields indicates depository fields and Blue fields indicate resorptive fields

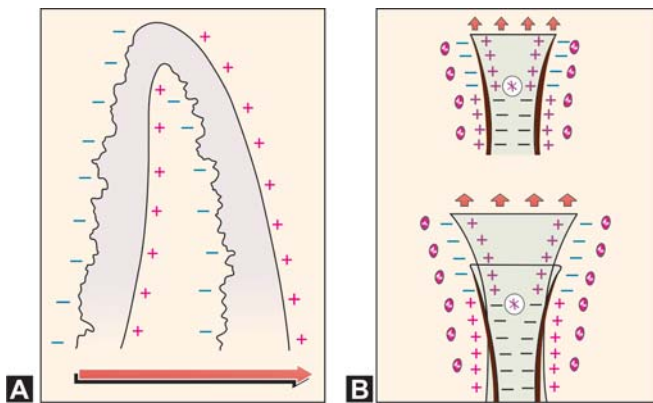
growth process are often termed growth sites. Mandibular condyle is one such example. But these growth sites do not contribute to the entire growth of the bone.

Some growth sites have been called "growth centers", a term which is applied to describe very active growth fields significant to the growth processes such as the cranial and facial sutures, the mandibular condyles, the nasal septal cartilage and synchondroses of the cranial base.

Endosteal and Periosteal Bone Growth

Approximately half of the cortical plate of the facial and cranial bones is formed by the outer surface, i.e. the periosteum, and the other half by the inner surface, the endosteum (Figs 4.6A and B).

Appositional layers of cortical bone can originate entirely from the periosteum or the endosteum. In some cases, the same cortex is composed of periosteal and endosteal bone layers which are separated by reversal lines (Figs 4.7A and B). This type of bone growth indicates that there has been a change in the direction of growth at some time. As new cortical bone is always deposited on the surface facing toward the direction of growth, bones revert to a type of periosteal bone formation from endosteal bone formation or vice versa. The reversal line represents the interface between endosteally and periosteally produced bone layers.



Figs 4.6A and B: (A) If the direction of the growth remains constant, the right cortical is formed periosteally and left formed endosteally. Both shift in unison in the direction of the growth. (B) The direction of bone can change during development of bone. In the area marked with an asterisk bone formation initially occurs endosteally (above) and at a later date after reversal of the direction of growth, periosteally (below)

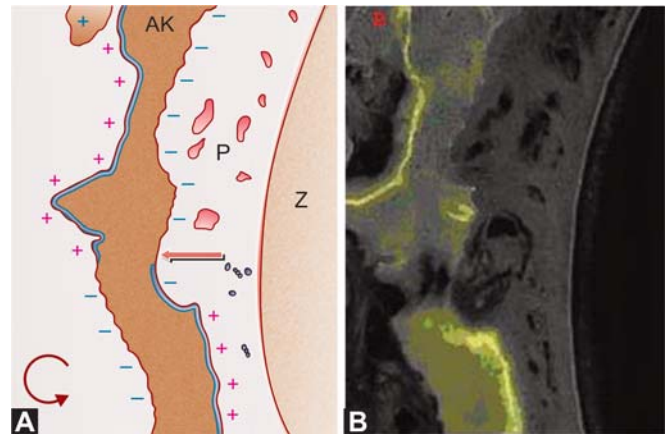
Remodeling

Facial bones undergo resizing and reshaping simultaneous to bone deposition and resorption. The reshaping of bone occurs not due to generalized deposition and resorption. Bone shaping requires differential growth activity, known as remodeling. Remodeling is a part of growth process, provides regional changes in shape, dimensions and proportions. It also provides regional adjustments that adapt to the developing function of the bone and its various growing soft tissues.

Types of Remodeling

There are four kinds of remodeling in bone tissues; *biochemical remodeling*: this involves continuous deposition and removal of ions to maintain mineral homeostasis; *growth remodeling*: the constant replacement of bone during childhood; *Haversian remodeling*: the secondary process of cortical reconstruction of bone as primarily vascular bone is replaced and *pathological remodeling*: regeneration and reconstruction of bone during and following pathology or trauma.

The reason why a bone should remodel is that its regional parts become moved. Drift moves each part of the bone as the bone enlarges. This calls for sequential



Figs 4.7A and B: (A) Reversal line: The interface between periosteally and endosteally formed bone is termed the reversal line. Line drawing of the histological section: AK—alveolar bone; P—periodontal space; Z—tooth root. (B) Section through an alveolar bone. The yellow staining shows endosteal bone formation in upper section of the surface facing the tooth and periosteal formation in the lower section. This leads to rotation of the bone structure (fluorescent microscopic view after tetracycline staining)

remodeling changes in shape and size of each region. The ramus, for example, moves posteriorly by a combination of deposition and resorption. As it does so, the anterior part of the ramus becomes remodeled into a new addition from the mandibular corpus. This progressive sequential movement of component part of the lengthening corpus becomes relocated into the area previously occupied by the ramus. Structural remodeling from what used to be part of the ramus into what then becomes a new part of the corpus takes place. Growth and remodeling are inseparable part of the same process. The same deposition and resorption that carry out the overall growth enlargement of whole bone carry out relocation and remodeling at the same time. Remodeling is paced by the soft tissues or the growth fields. The functions of remodeling include to progressively enlarge each whole bone, to sequentially relocate each of the component parts of the whole bone to allow for overall enlargement, to shape the bone to accommodate its various functions in accordance with the physiologic actions exerted on that bone and carry out regional structural adjustments so that functional fitting of all the separate bones to each other and to their soft tissues is achieved (Fig. 4.8).

GROWTH MOVEMENTS

Drift and Displacement

Two kinds of growth movements, namely cortical drift and displacement are seen. All bones have one common growth principle, that is drift, which was termed by Enlow

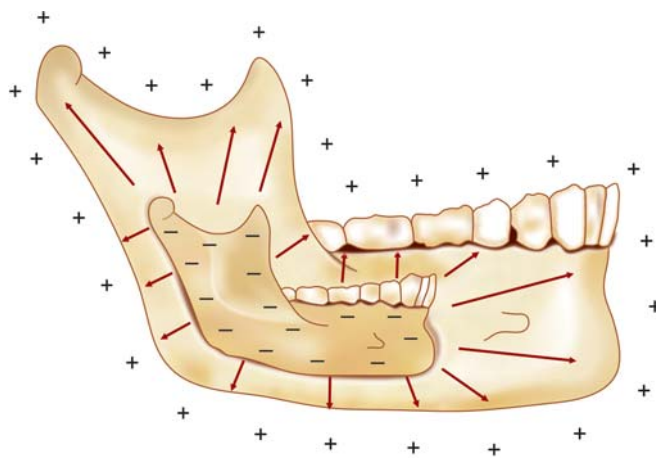


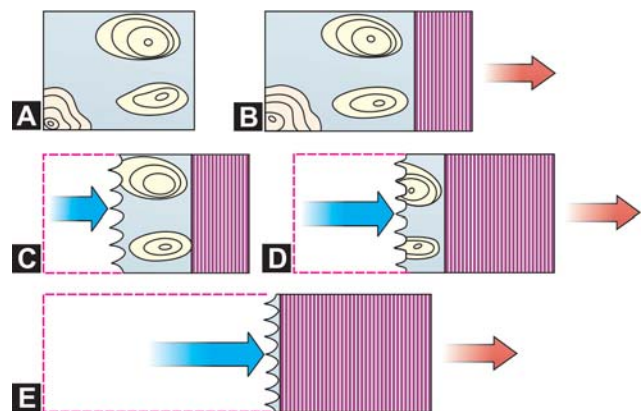
Fig. 4.8: Remodeling involves constant resizing and reshaping of the bone. The basic shape of bone is maintained

(1963). *Drift* is growth movement (relocation or shifting) of an enlarging portion of a bone by the remodeling action of its osteogenic tissues, while *displacement* is a physical movement of a whole bone.

The cortical plate can be relocated by simultaneous apposition and resorption processes on the opposing periosteal and endosteal surfaces (cortical drift). The bony cortical plate drifts by depositing and resorbing bone substance on the outer and inner surfaces respectively, in the direction of growth (Figs 4.9A to E). If resorption and deposition take place at the same rate, the thickness of the bone remains constant. Should more bone be deposited than resorbed, the thickness of the structure increases. During the developmental period, deposition takes place at a slightly faster rate than resorption, so that the individual bones slowly enlarge. The teeth follow the drift of the alveolae while the jaw is growing and thus maintain their position within the surrounding bony structures despite the bone displacement.

Displacement

Displacement is movement of the whole bone as a unit. It is a translatory movement of the whole bone caused by the surrounding physical forces, and is the second characteristic mechanism of skull growth. The entire bone



Figs 4.9A to E: Process of cortical drift: (A) Cortical plate of bone; (B) increase in thickness due to apposition on one of the surfaces; (C) When the resorption process on one side of the bone exceeds the apposition process on the opposing side, the thickness of the bone will be reduced; (D) When resorption on one side of the bone corresponds in magnitude to apposition on the opposing side, the bone will drift without changing its size; (E) The cortical plate has drifted completely to the right when compared to its original position in 'A' by the process of remodeling

is carried away from its articular interfaces (sutures, synchondroses, condyle) with adjacent bones. Displacement is of two types namely primary displacement and secondary displacement.

Primary displacement: As a bone enlarges, it is simultaneously carried away from the other bones in direct contact with it. This creates space within which bony enlargement takes place. This is termed as primary displacement. It is the physical movement of the whole bone, as the bone grows and remodels by resorption and apposition (Fig. 4.10).

Secondary displacement: It is the movement of a whole bone caused by the separate enlargement of other bones, which may be nearby or quite distant. It is the movement of bone related to enlargement of other bones. For example, growth in the middle cranial fossa results in the movement of the maxillary complex anteriorly and inferiorly (Fig. 4.11).

Drift and displacement occur together and complement each other. It is very difficult to determine the separate contributions of drift and displacement to the remodeling of bone.

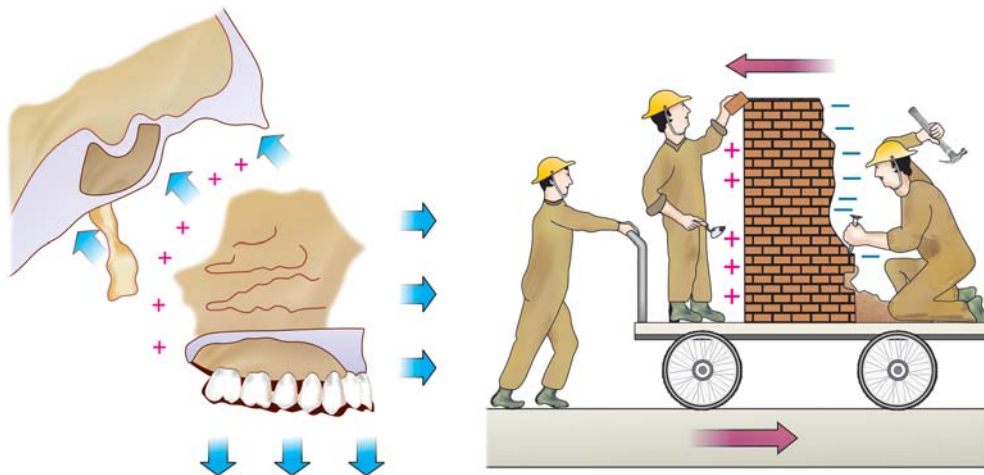


Fig. 4.10: Primary displacement. Bone moves from one position to another not only because of deposition and resorption but also because of space created by enlarging bones

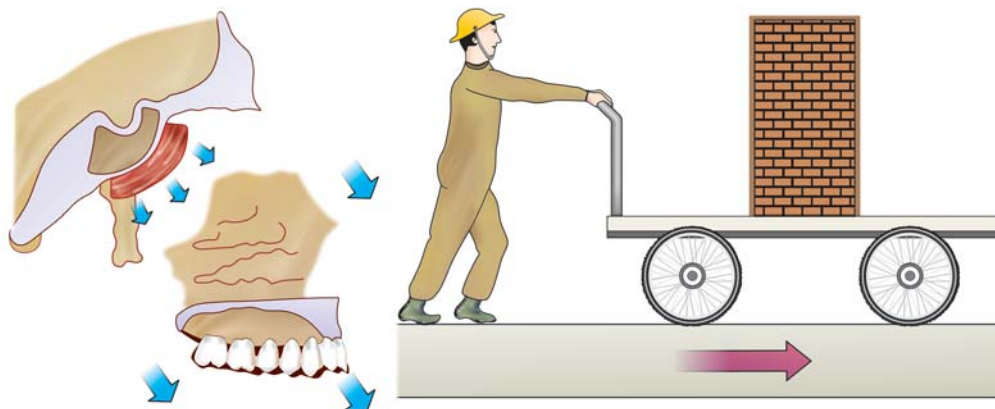


Fig. 4.11: Secondary displacement. Bone moves from one position to another not because of deposition and resorption but because of surrounding physical forces

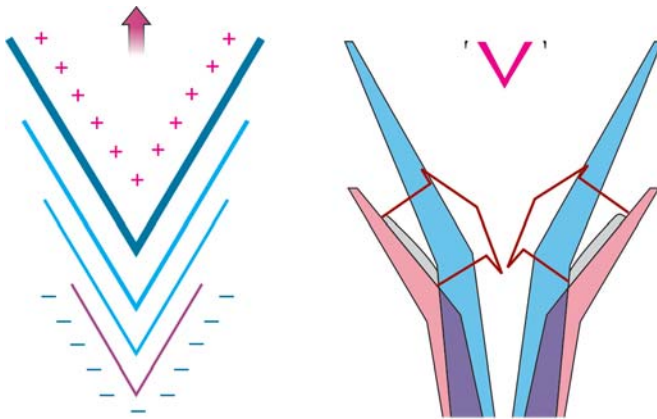


Fig. 4.12: Expanding V principle—vertical expansion. Bone is deposited on the inner surface of 'V' shaped bone and resorbed on the outer surface. Thus, the 'V' moves away from its narrow end (direction of the arrow) and enlarges in overall size

V Principle

The V principle is an important facial skeleton growth mechanism, since many facial and cranial bones have 'V' configuration or 'V' shaped regions. The areas grow by bone deposition on the inner side due to the concept of surface growth depending on growth direction. Resorption takes place on the external surface of the 'V'. The 'V' moves away from its tip and enlarges simultaneously. Thus an increase in size and growth movement takes place in a unified process. Hence it is also called expanding 'V' principle. The movement of the bone is towards the broad end of the 'V' (Fig. 4.12).

Longitudinal section through the right and left coronoid processes of a mandible reveals that the processes are enlarged during growth. In accordance with the 'V' principle, bone is deposited on the lingual surfaces and resorbed from the opposing buccal surfaces. The structures increase in height, the tips of the coronoid processes diverge further, and their bony bases converge (Fig. 4.13).

Surface Principle (Fig. 4.14)

The surface principle states that bone sides which face the direction of the growth are subject to deposition and those opposed to it undergo resorption. These processes always take place on contralateral bone surface so that the cortical plate follows the course of growth. The

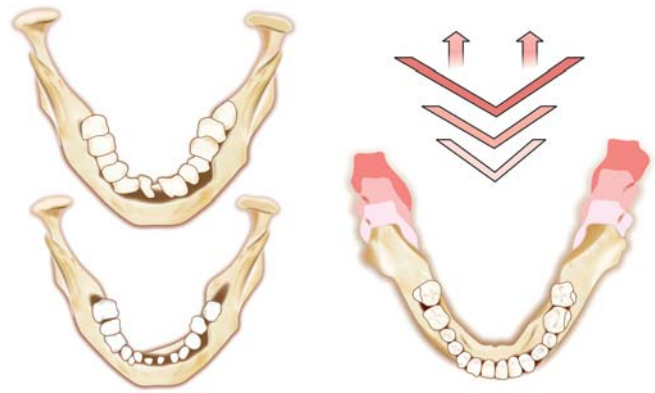


Fig. 4.13: The 'V' principle—horizontal expansion. Mandibular configuration of a five year old and an adult viewed from above. The mandible is viewed from above, including a horizontal section through the base of the coronoid process. Bone is deposited on the lingual side of the mandibular structures up to the ramal surface. Thus, the coronoid process move—despite bone deposition on the inner surfaces in backward direction and the posterior parts of the mandible widen (Enlow 1982)

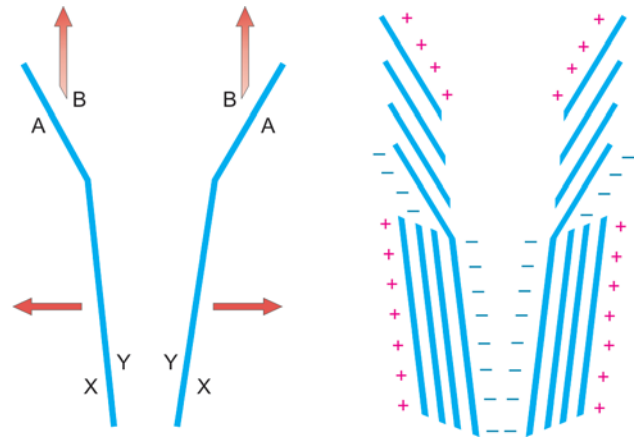


Fig. 4.14: Surface principle. The areas marked 'X' on the outer surface of the bone and those marked 'B' on the inner surfaces are in the direction of growth and are depositor. Accordingly, areas 'A' and 'Y' resorb in the opposite direction

direction of growth is not the same for all areas of the bone as each region of a structure has its own specific growth pattern. Reversals in the direction of growth can result in bone deposition and resorption processes taking place directly adjacent to one another on the same cortex. As individual parts of the bone grow in different

directions, only half of the deposition process is localized in on the outer cortical plate (periosteal bone formation). The other half of the growth process consists of bone deposition on the inner cortical surface (endosteal bone formation).

Posterior Growth and Anterior Displacement

The overall growth pattern of maxilla and mandible can be explained in two different ways. If the cranium is considered as the reference area, the maxilla and mandible moves downward and forward. On the contrary, findings from vital studies have shown particularly in the mandible the posterior surface of the ramus, the condylar and coronoid processes are the principal sites of growth with little changes along the anterior part of the mandible. This proves the concept that the jaw bones are translated downward and forward while it grows upward and backward in response to the translation. This helps to maintain spatial contact with the skull (Figs 4.15 and 4.16A and B).

GROWTH EQUIVALENTS CONCEPT/ENLOW COUNTERPART PRINCIPLE

This is a concept in which the certain facial and cranial parts are compared with each other to see how they fit. The vertical or horizontal size of one given part is compared with its specific counterparts. A dimensional balance exists if both of them match. Imbalances can result in either protrusion or retrusion of that part of the face.

According to Enlow, the growth activity in one region is invariably accompanied by complementary growth in other regions. This complementary activity is essential for maintaining functional and esthetic balance. Enlow pointed out, both the dimensions and alignment of the craniofacial components are important in determining the overall facial balance. Thus if the anterior facial height is long, facial balance is preserved if the posterior facial height and mandibular ramus height are also relatively large. On the other hand, short posterior facial height can lead to a skeletal open bite tendency and disturbance in facial proportionality. Similarly, alignment would affect the vertical and anteroposterior position of the various skeletal units and could compensate or worsen a tendency toward imbalance. For example, if maxilla were rotated down posteriorly, a long ramus and acute gonial angle

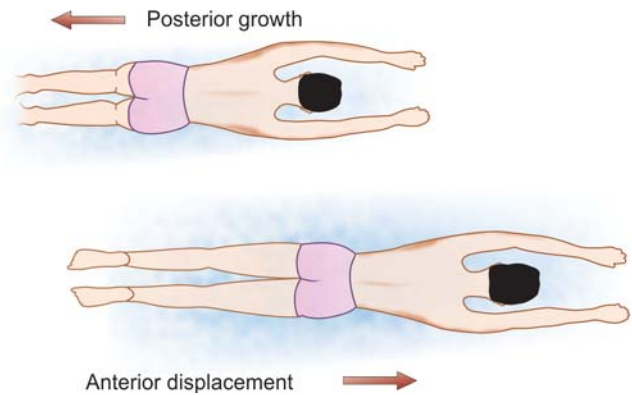
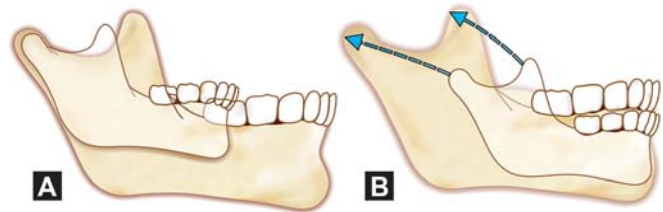


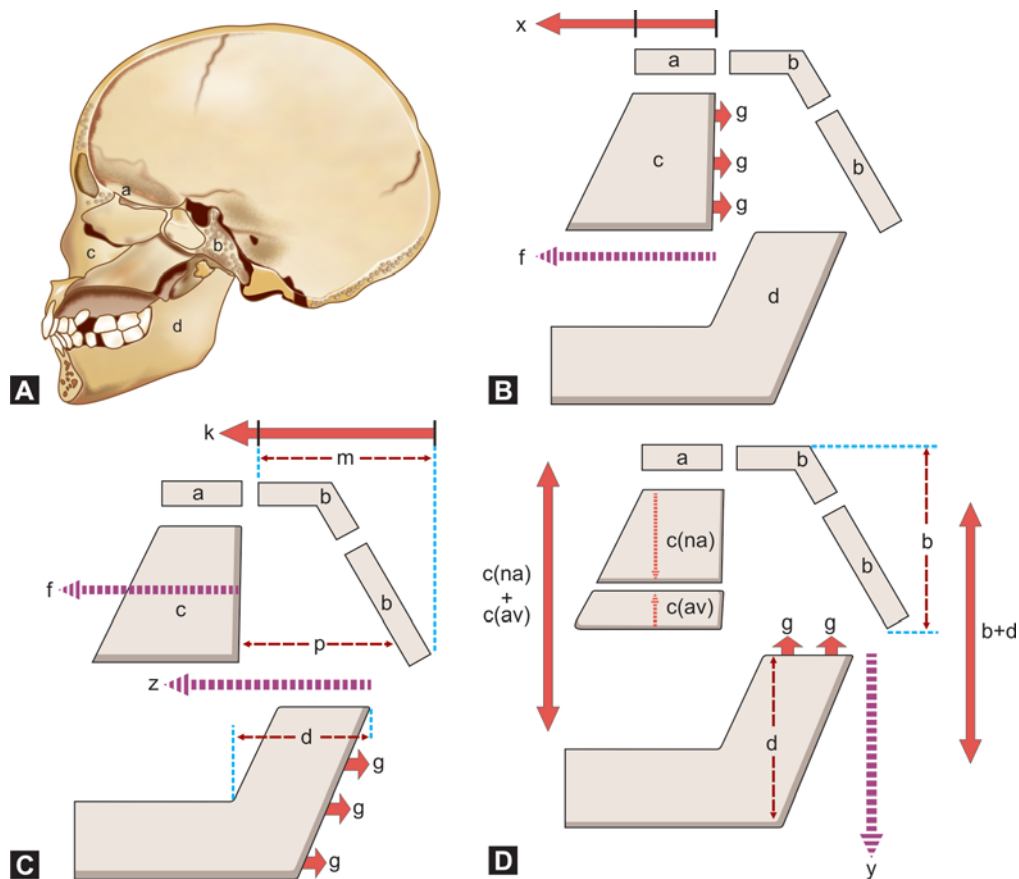
Fig. 4.15: Comparison of posterior growth—anterior displacement with a swimmer who dives from the board



Figs 4.16A and B: (A) Mandible grows downward and forward if cranial base is taken as reference and (B) Vital studies have shown that the concept B is correct and the mandible grows backwards and upwards

would compensate and allow normal facial proportions, but even a slightly short ramus would produce downward-backward mandibular rotation and a long face-open bite tendency. Thus, Enlow stresses the importance of complementary growth of facial skeleton to preserve the facial harmony. Based on this concept of growth equivalents, Enlow introduced counterpart analysis to assess the rotation of jaw bases.

Different counterparts or Growth equivalents (Figs 4.17A to D): Nasomaxillary complex elongation is the counterpart for elongation of anterior cranial fossa. Lengthening of spheno-occipital region is the growth equivalent of the underlying pharyngeal region and the increasing length of ramus. Combined vertical lengthening of the clivus and mandibular ramus is the growth equivalent of total vertical nasomaxillary region. Maxilla and mandible corpus are mutual counterparts.



Figs 4.17A to D: Growth equivalents concept of Enlow. (A) Components of craniofacial region (a= anterior cranial base; b= spheno-occipital synchondrosis; c= nasomaxillary complex; d= mandible) (B) Elongation of anterior cranial base (a) causes simultaneous enlargement of nasomaxillary complex(c). (C) Lengthening of spheno-occipital region (m) is the growth equivalent for underlying pharyngeal region (p) and increasing length of ramus distance (d). These growth equivalents cause normal positioning of mandible relative to nasomaxillary complex. (D) Combined vertical lengthening of clivus (b) and mandibular ramus (d) is the growth equivalent for the total vertical elongation of nasomaxillary region

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Chapter

5

Control Mechanisms in Craniofacial Growth

CHAPTER OUTLINE

- **Changing Paradigms of Craniofacial Biology**
 - Genomic paradigm
 - Functional paradigm
- **Site vs Center**
- **Controlling Factors in Craniofacial Growth**
 - Von Limborgh's classification
 - Enlow and Moyer's classification
 - Goose and Appleton's classification
- **Theories of Bone Growth**
 - Bone remodeling theory
 - Genetic theory
 - Sutural theory
 - Cartilaginous theory
 - Functional matrix theory
 - Servo system theory
 - Rate limiting ratchet hypothesis
 - Growth relativity hypothesis

The field of dentofacial orthopedics has been credited with number of competing and credible treatment methods. Most of the treatment approaches have been based on the fundamental biological mechanisms involved in the growth and development of craniofacial bones and teeth. The exact mechanism which controls the craniofacial growth has been a matter of debate and research for years together. From time to time, attempts have been made to provide an overriding conceptual framework for all craniofacial growth. Sometimes synthesis of several theories have been put forward.

Grasping or understanding of the concepts of craniofacial growth is of paramount importance for the orthodontist to understand the basic concepts of their discipline.

CHANGING PARADIGMS OF CRANIOFACIAL BIOLOGY

"Paradigm" is a Greek word which means the current conceptual framework of a scientific field. It is closely related to the normal science. Kuhn (1970) defines normal science as research that the members of a specific group of scientists recognize as central to their field. Paradigm on the other hand encompasses theories, hypotheses and facts (Fig. 5.1).

A single paradigm may be the basis for many theories and hypotheses. The evolution of various concepts or theories of growth can be studied under two different paradigms, namely "Genomic paradigm" and "Functional paradigm". A change in paradigm is brought about due to inconsistencies within the old paradigm or scheme.

The Genomic paradigm viewed craniofacial growth as primarily genetically predetermined and immutable. Brodie after noting the persistent pattern of facial configuration, assumed it was under tight genetic control. Numerous researches were focused on growth sites for this control; the sutures, cartilages of cranium and face,

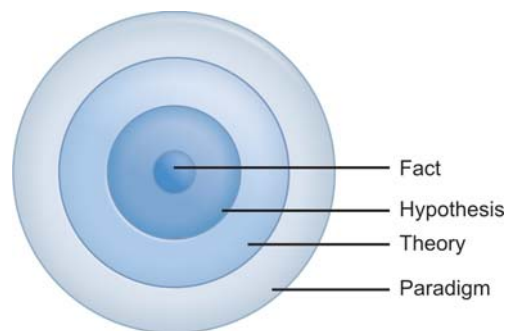


Fig. 5.1: Concept of paradigm

and periosteum. It was assumed that the cartilages and facial sutures were under tight genetic control and the brain determines the vault dimension.

Subsequent to the use of animals in craniofacial research, changed idea about genetic concepts emerged. Moss in 1960s extended the concepts of Vander Klaauw into the functional matrix hypothesis, which marked the beginning of functional paradigm. The functional matrix theory was a radical change into a new way of looking at craniofacial growth.

The functional paradigm emphasized the plasticity of growth and development of craniofacial skeleton. According to the functional paradigm, epigenetic interaction of intrinsic and extrinsic factors results in variation in craniofacial form. The principles of functional paradigm supported consideration of the use of dentofacial orthopedic technique in correcting the developing dentofacial malocclusion and deformity.

David S Calson has classified the changing paradigm into three eras:

1. 1920 to 1940
2. 1940 to 1960
3. 1960 to 1980.

1920 to 1940

Craniofacial research from 1920 to 1940 was based primarily on the study of the structure of the craniofacial skeleton. Little consideration was given for function. Krogman (1974) stated that during this period there was an essentially static approach to craniofacial research. Moss (1982) subdivided this phase into 1. "Preradiologic phase", where craniometry was widely relied upon and 2. "Radiologic phase". Radiologic phase starts with the advent of radiographic cephalometry. Early concepts about the craniofacial growth were extracted mainly from studies of comparative anatomy, craniometrics and radiographic cephalometrics. It was assumed during this period that growth of the craniofacial skeleton was largely genetically predetermined and immutable. Moss has described the general belief during this period into a classic triad:

- That sutures are the primary growth sites.
- That growth of the cranial vault occurs by periosteal deposition and endosteal resorption.
- That all the cephalic cartilages including the cranial base, nasal septum and mandibular condyle are primary growth "centers" under direct genetic control.

This period of craniofacial biology is called as genomic paradigm.

1940 to 1960 (Fig. 5.2)

Craniofacial biology during this period marked the beginning of introduction of concepts of function and adaptation to altered function.

There was an increased emphasis on experimental animal research in an effort to find out the actual mechanism of facial growth. During this period, there was development and use of technologies like radio opaque implants, vital dyes, autoradiography and *in vivo* and *in vitro* transplantation.

Moss's experimental analysis of sutural growth, established beyond doubt, that, sutures within cranial vault and face are sites of active, but compensatory skeletal growth. This led to a tangible shift in the thought of craniofacial biologists. Moss also established that sutural growth and the form of the individual bones of the vault are not genetically determined (Fig. 5.3). Questions were beginning to be raised about the validity of the genomic paradigm.

Krogman has described two approaches by the end of 1950s within the single dominant genomic paradigm of craniofacial biology, the "comprehensive approach" and "stucturofunctional approach". Comprehensive approach continued the earlier tradition of craniometrics with radiographs, cephalostat, while the stucturo-functional approach focused primarily on the cause and effect relationships in the craniofacial complex.

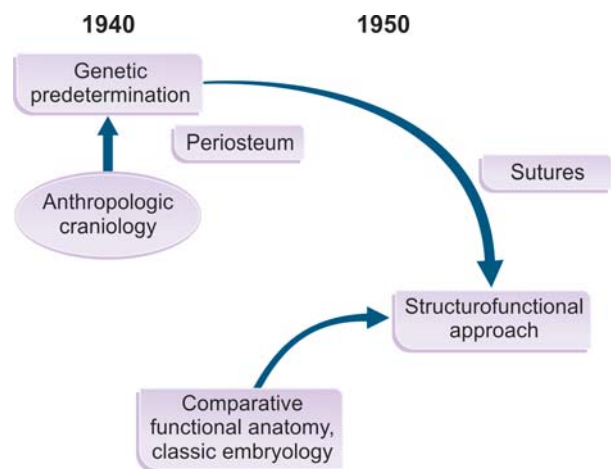


Fig. 5.2: Model of craniofacial biology during 1940s and 1950s

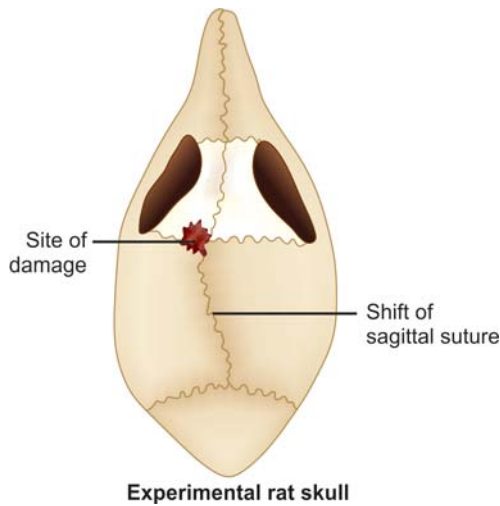


Fig. 5.3: Classic experiment by Moss: Cautery of cranial suture resulted in shift of the suture which questioned the genetic potential of sutures

Periosteal and sutural bone growths were no more considered to have tight genetic control and were removed from genomic paradigm. But there was little evidence at the time against the cephalic cartilages and condyle. Therefore, genomic paradigm continued to dominate. The alternate view that "function" plays a vital role during craniofacial growth continued to gather momentum.

1960 to 1980 (Fig. 5.4)

This period marked the foundation of alternative paradigm and also the beginning of scientific revolution

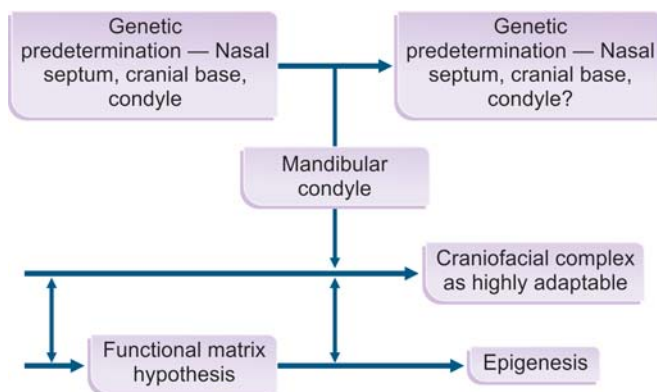


Fig. 5.4: Model of craniofacial biology during 1960s and 1970s

in craniofacial growth. This period in fact belonged to one individual namely, Melvin Moss. His "functional matrix hypothesis" is in fact considered to be the alternate paradigm itself. Functional matrix hypothesis has been the subject of debate, experimental analysis and discussion than any other theory. The emphasis is on that all skeletal tissues are responsive and having a degree of plasticity during growth and development. This alternative paradigm called as the "functional paradigm" states that the craniofacial complex is highly adaptable, both ontogenetically and phylogenetically to the functional demands placed on it and to its developmental environment. The functional paradigm maintains that the burden of proof lies with those who believe that there is any genetic influence on craniofacial skeletal growth. Moss stresses more on the epigenetic hypothesis, which states that "both structure and function evolve alterations in the biomechanical, biophysical, biochemical and bioelectric parameters of the developing organism, both intra and extracellularly". These alterations of state act significantly to regulate subsequent developmental stages as well as to regulate genomic reaction to these altered environmental state. In this hypothesis, *environment is not just permissive and supportive but also regulative*.

Future of Craniofacial Biology

With clinical orthodontics in mind, the field of craniofacial biology has mainly emphasized research on postnatal craniofacial growth from birth through skeletal maturity. Dentofacial orthopedics might be attempted during this period only. Now the focus is equally aimed at developmental biology also, which includes areas of heredity, genetics and embryology. The role of neural crest cells in craniofacial development and of the factors that influence their migration and differentiation have become areas of interest. More research is also done on teratology and the effects of teratogen on the development. Discovery and identification of specific regulatory factors like growth factors and homeobox genes have thrown more light on the morphogenesis and development of craniofacial complex.

In the present era, there are three issues which are important to craniofacial growth and development. They are:

1. There are a number of genetically encoded regulatory factors that have profound effects on the

morphogenesis and prenatal development of craniofacial complex.

2. All these factors operate within the epigenetic milieu from the level of position of genes on the chromosome to the interaction of cells and entire organisms with the external environment.
3. There is ample evidence to support and conclude that morphogenesis, prenatal development and postnatal growth of the craniofacial complex can be modified in a predictable, controlled and clinically effective way.

Probably within the next few decades, orthodontists will be using molecular kits to diagnose growth related problems and determine precisely each patient's developmental status as well as the presence or absence of growth factors and signaling molecules.

SITE VS CENTER

A proper understanding of the terms growth site and growth center will help to clarify the differences between theories of growth. Baume had coined these two terminologies. According to him, “growth centers” are places of endochondral ossification with tissue separating force, contributing to the increase in skeletal mass. Growth site has been defined as a region of periosteal or sutural bone formation and modeling resorption adaptive to environmental influences. Profitt defines growth site as merely a location at which growth occurs whereas center is a location at which independent or genetically controlled growth occurs. *All growth centers are also sites, whereas all growth sites are not centers.* Most of the theories of growth are based on where the growth center is expressed.

Enlow and Moyers use a common term *growth fields* which includes both growth sites and centers. All surfaces of bone are covered by an irregular pattern of *growth fields* comprising of various soft tissue osteogenic membranes or cartilages. *Bone does not grow by itself, instead it is grown by the environment.* Growth “sites” are growth fields having special roles in the growth of particular parts of bone. Examples of growth sites include mandibular condyle, maxillary tuberosity, synchondroses, sutures, alveolar process, etc. They do not cause growth of the whole bone. *Growth center* implies special areas which control the overall growth of bone. These growth centers have “force” or “energy” within them for bone growth.

CONTROLLING FACTORS IN CRANIOFACIAL GROWTH

There are different methods of classifying the controlling factors.

Von Limborgh's Classification (Table 5.1)

- Intrinsic genetic factors
- Local genetic factors
- General epigenetic factors
- Local environmental factors
- General environmental factors

Enlow and Moyers' Classification

Natural

- Genetic
- Function
- General body growth
- Neurotrophism

Table 5.1: Von Limborgh's classification

S. No.	Factor	Definition/Explanation
1.	Intrinsic genetic factors	Genetic factors inherent to the craniofacial skeletal tissues.
2.	Local epigenetic factors (capsular matrix)	Genetically determined influences originating from adjacent structures and spaces (brain, eyes, etc.).
3.	General epigenetic factors	Genetically determined influences originating from distant structures (sex hormones).
4.	Local environmental factors (periosteal matrix)	Local nongenetic influences from external environment (Muscle force, local external pressure).
5.	General environmental factors	General nongenetic influences originating from the external environment (oxygen supply, food).

Disruptive Factors

- Orthodontic forces
- Surgery
- Malnutrition
- Malfunction
- Gross craniofacial anomalies

Goose and Appleton's Classification

- Endocrinal factors
- Multifactorial inheritance
- Racial differences
- Nutrition
- Diseases
- Socioeconomic factors
- Secular trends

Genetics

Morphogenesis of the bones and cartilage of the craniofacial skeleton initiated during embryogenesis continues through childhood and is completed during adulthood. It is generally thought that the bone growth at the cranial and facial suture occurs when the bone is gradually forced apart by underlying tissues such as the brain, dura mater and nasal cartilages which exert tensile force at the growth sites. But even this has an underlying intrinsic or genetic component as there is stimulation of growth factors and transcription factors.

Cartilage: The molecules and molecular signaling cascades involved in the regulation of cartilage growth have been deciphered as follows:

Parathyroid hormone related peptide (PTHrP), stimulates chondrocyte proliferation and inhibits chondrocyte hypertrophy. Indian hedgehog (Ihh) controls both chondrocyte proliferation and hypertrophy

through molecule circuitry with PTHrP and parathyroid hormone receptors (PTHrR). Bone morphogenetic proteins (BMPs) stimulates chondrocyte differentiation, hypertrophy and mineralization.

Fibroblast growth factors (Fgf), signaling through fibroblast growth factors receptors (Fgfr) inhibit chondrocyte proliferation. Transforming growth factor beta (Tgf- β) stimulates chondrocyte differentiation. It also plays a role in inhibiting chondrocyte proliferation. Table 5.2 gives the possible role of various growth factors in endochondral growth.

Sutural Growth

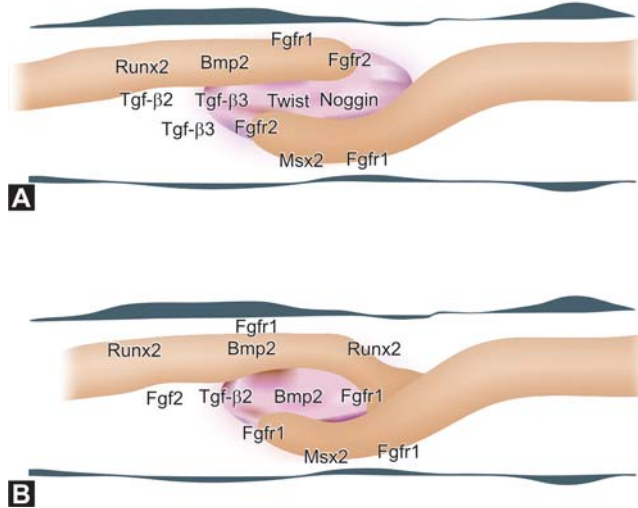
Craniofacial suture morphogenesis and maintenance of sutures as potent bone growth sites are regulated by tissue interaction with the underlying duramater (Figs 5.5A and B). This regulation results from secretion of soluble factors by the duramater, presumably in response to growth signals from the expanding, underlying neurocranium. Craniofacial synostosis or premature obliteration of sutures results in abnormal morphogenesis throughout the head.

Studies have shown that growth factors such as transforming growth factors beta 1 (Tgf- β 1), Tgf- β 2, Tgf- β 3, bone morphogenetic protein 2 (BMP 2), BMP 7, fibroblast growth factor 4 (Fgf4), insulin like growth factor 1 (Igf-1) and sonic hedgehog (Shh) are found in the sutures and duramater. Addition of Tgf- β 2 and Fgf4 to rat is found to induce synostosis. Over expression of transcription factor induces suture obliteration.

Mutation in genes for fibroblast growth factor receptors 1, 2 and 3 (Fgfr1, Fgfr2 and Fgfr3) is associated with craniofacial synostosis in humans. It is generally believed that facial sutures function in the same manner as cranial suture during craniofacial bone growth.

Table 5.2: Genetic growth factors and their possible role in endochondral growth

Genetic Factor	Function
Parathyroid hormone related peptide {PTHrP}	Stimulates chondrocyte proliferation, inhibits chondrocyte hypertrophy.
Indian hedgehog {Ihh}	controls chondrocyte proliferation and hypertrophy.
Bone morphogenetic proteins {BMPS}	Stimulates chondrocyte differentiation, hypertrophy and mineralization.
Fibroblast growth factors {Fgf}	Inhibits chondrocyte proliferation.
Transforming growth factor beta {Tgf- β }	Stimulates chondrocyte differentiation, inhibits chondrocyte proliferation, hypertrophy and mineralization.
Hyaluronan and CD 44	Controls lengthening of cranial base.



Figs 5.5A and B: Diagrams showing distribution of growth and transcription factors in sutures. (A) Open suture (B) Fusing suture (Source: Seminars in orthodontics December 2005)

Multifactorial Interaction

There is usually no characteristic expression of typical Mendelian ratios in the offspring. In Multifactorial inheritance, the likeness of parents and offspring can be expressed by correlation coefficients. Correlation coefficient can take any value between +1 to -1. +1 implies perfect correlation and, -1 means negative correlation and 0 implies no correlation or relationship.

Presence of recessive gene with additive effects results in a correlation coefficient of 0.5 between parents and offspring. Marriage between couples with similar characteristics results in a higher correlation coefficient. Examples for Multifactorial inheritance include achondroplasia, Marfan's syndrome.

Function

Role of normal function is essential for normal growth and development of skeleton or bones. This is the essence of functional matrix hypothesis of Moss. Lack of function results in retarded growth in relation to that area. Classical examples include condition like microglossia, TMJ ankylosis, and muscle dysfunction.

General Body Growth

General body growth (somatic growth) and craniofacial growth are interrelated, but this relationship cannot be

precisely predicted. Considerable amount of studies have been done to find out the relationship between maturation of somatic growth and dimensional growth in craniofacial region. Majority of the studies have concentrated on the prediction of maximum height velocity. This is done to predict the timing of peak height velocity which could be utilized for orthopedic correction. Skeletal age derived from hand wrist radiographs is based on this principle.

Neurotrophism

Neurotrophism has been defined as "the nonimpulsive transmissive neurofunction involving axoplasmic transport, providing for the long term interactions between neurons and innervated tissue, which homeostatically regulate the morphological, compositional and functional integrity of those tissues". The neural control of skeletal growth is assumed to be by transmission of substances or transmitters. Neurotrophism could act either directly or indirectly. In direct control, there could be osteogenesis by direct neural stimulation. In indirect, nerves control or modulate skeletal growth through soft tissue stimulation. Direct effect has not been demonstrated experimentally. Neurotrophism has been dealt with extensively along with functional matrix theory of bone growth.

Disruptive Factors

These factors do not contribute to variations, but they can induce changes in growth. Examples include:

1. Orthodontic/orthopedic forces when used to alter growth.
2. Surgical procedures interfering with growth.
3. Malnutrition.
4. Malfunction: altered nasal respiration (Linder Aronson et al), and posture (Beni Solow and Tallgren) are found to produce changes in craniofacial growth.
5. Gross craniofacial anomalies like synostosis alter the normal growth.

Endocrine Control

Growth hormones produced by pituitary gland are essential for normal growth. Growth hormones also stimulate the production of a further group of hormones called somatomedin. Somatomedins are mainly produced in liver. Somatomedin act on growing cartilage cells (Fig. 5.6).

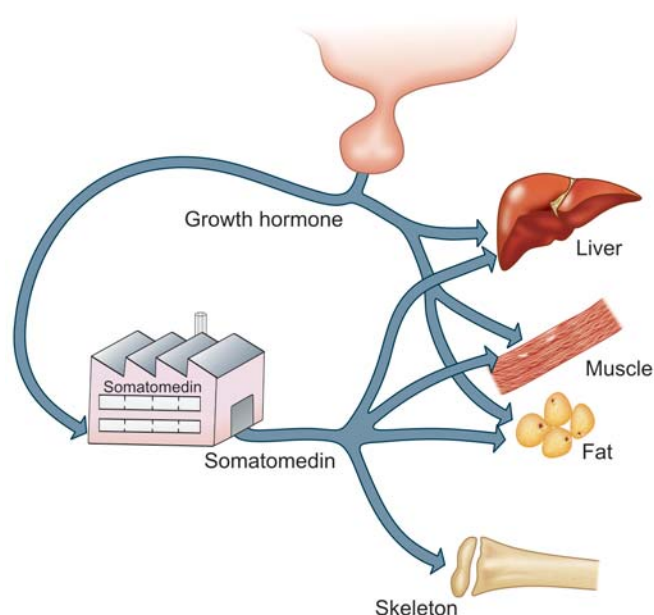


Fig. 5.6: Schematic representation of direct and indirect control of growth hormone through somatomedin

Thyroxin is essential for development in early fetal life, nervous system development and also for general growth. Deficiency of thyroxin in early life affects the brain and also growth whereas in late life causes slow growth and delayed maturity.

Puberty or adolescence is the result of interplay of various hormones like follicle stimulating hormone (FSH) and luteinizing hormone (LH). They produce estrogen in females and testosterone in males. Estrogen causes growth of breasts, vagina, uterus and eventually menstruation in girls. Testosterone produced due to the influence of luteinizing hormone causes growth of penis, prostate and seminal vesicle. FSH is involved in testicular growth and spermatogenesis. Growth hormones and androgens play crucial role in the onset of adolescent growth spurt.

Racial Differences

There is difference in the growth among various races. Asians are generally shorter than Europeans. Differences in body proportions also exist (Fig. 5.7).

Nutrition

Malnutrition causes retarded as well as delayed growth in children.

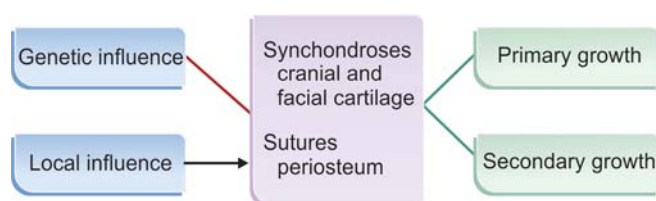


Fig. 5.7: Compilation of important growth controlling factors

Diseases

Minor diseases in normal children does not have any effect on growth, but is found to alter the mineralization of teeth. But in a malnourished child, minor diseases do play a negative role in affecting growth. Chronic debilitating illness has a profound influence in affecting the growth. Recently even psychological factor has been shown to affect the growth status of individuals.

Socioeconomic Status

Children from different social groups display variation in size and speed of their growth. Children who belong to lower strata have delayed growth. Family size also has an effect on growth in lower socioeconomic groups. Eruption of teeth and onset of menarche is earlier in socially higher class of population.

Secular Trend

With evolution, there is a trend towards children growing larger than their parents and also maturing earlier. Better nutrition and fewer occurrences of diseases in childhood due to immunization could be the reason for this change, but the exact mechanism is not fully understood.

THEORIES OF BONE GROWTH

Attempts have been made for quite sometime to provide a basis or framework for craniofacial growth in the form of theories, concepts or hypotheses. Most of these efforts have met with criticism or failure because of the varied and complex nature of craniofacial growth. The first scientific research on craniofacial growth has been credited to Sir John Hunter in the 18th century for his studies on growth of the jaws and eruption of the dentition. The theories are based on the fact where the

intrinsic genetic potential or growth center is expressed. The various theories of growth are:

- Bone remodeling theory
- Genetic theory
- Sutural hypothesis
- Cartilaginous theory
- Functional matrix theory
- Servo system theory
- Composite hypothesis by von Limborgh
- Rate limiting ratchet hypothesis
- Growth relativity hypothesis.

Bone Remodeling Theory of Craniofacial Growth by Brash (1930)

Introduction of vital staining method by John Hunter helped Brash to postulate the first general theory the "bone remodeling theory". This theory concluded that bone grows only by interstitial growth. The three fundamental tenets of this theory are:

1. Bone grows only by apposition at the surfaces.
2. Growth of jaws takes place by deposition of bone at the posterior surfaces of the maxilla and mandible. This is described as "Hunterian growth".
3. Calvarium grows through bone deposition on the ectocranial surface of the cranial vault and resorption of bone on the endocranial surface (Fig. 5.8).

Bone remodeling theory postulated that the craniofacial skeletal growth takes place by bone remodeling—selective deposition and resorption of bone at its surfaces.

The Genetic Theory (A. Brodie—1941)

The genetic theory simply stated that genes determine and control the whole process of craniofacial growth. But the mechanism of action by the genetic unit and the mechanism by which the traits are transmitted were not understood until recently. Gregor Mendel (1822-1884) opened up the field of genetics, notably regarding the mechanism of inheritance and transmission.

The field of genetics consists of two principle areas of interest:

1. "Transmission genetics" is characterized by statistical approach and involved only in explaining possible method of transmission. It is based on Mendelian laws and did not explain about genes or its characteristics. Weisman in late 19th century introduced the concept of "germ plasm". As per this

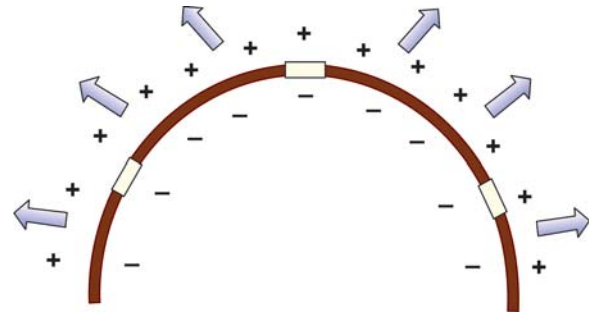


Fig. 5.8: Diagrammatic representation of the remodeling theory of craniofacial growth using the cranial vault as a model. Increase in the size of the cranial vault occurs by adding bone via periosteal deposition on the outer, ectocranial surface and resorption of bone on the inner, endocranial surface of the vault (*Source: Seminars in orthodontics December 2005*)

idea, the determinant of traits that is transmitted from parents to offspring is present in the cytoplasm of the gametes. Mendel introduced the term "Pangene" to describe the germ plasm. In the year 1909, Bateson introduced the term "genetics" following which Johnson used the term "gene" to the presumed unit of heredity. Transmission genetics could not explain all the changes taking place in craniofacial growth. As the genetic theory failed to explain so many occurrences on craniofacial growth, the focus shifted from transmission genetics to molecular genetics.

2. Developmental and molecular genetics. This field has undergone profound development and discoveries following extensive research.

The Sutural Hypothesis/Sutural Dominance Theory (Sicher and Weinman)—1952

Sicher and Weinman, two great anatomists, introduced the sutural hypothesis. According to this theory, sutures, cartilages and periosteum are all responsible for facial growth and were assumed to be under intrinsic genetic control. Sicher came to the conclusion that sutures were causing most of the growth based on the studies using vital dyes.

Essence of the Theory (Fig. 5.9)

According to Sicher, the sutures are the primary determinants of craniofacial growth. The craniofacial skeleton enlarges due to the expansive forces exerted by the sutures as they separate.

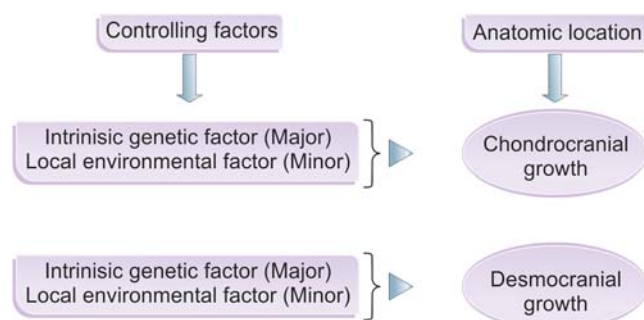


Fig. 5.9: Controlling factors of growth according to Sicher

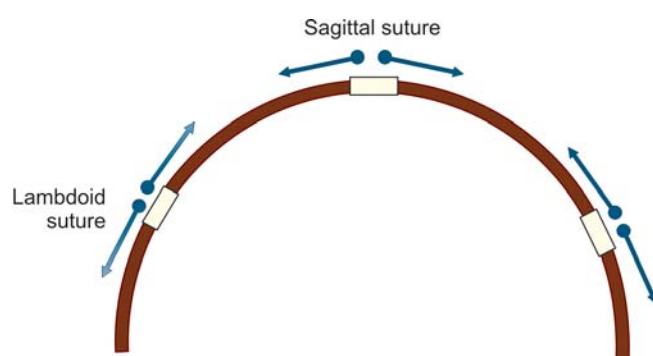


Fig. 5.10: Diagrammatic representation of the sutural theory of craniofacial growth using the cranial vault as a model. Increase in the size of the cranial vault takes place via primary growth of bone at the sutures, which forces the bones of the vault away from each other (*Source: Seminars in orthodontics December 2005*)

Theory (Fig. 5.10)

Sicher stated that all bone forming elements like sutures, cartilage and periosteum are growth centers like the epiphysis of long bones. Though Sicher held cartilage, sutures and periosteum as responsible for facial growth, this theory is called "sutural dominance theory" because he believed that the primary event in sutural growth is the proliferation of the connective tissue between the two bones. Proliferation of the sutural connective tissue creates the space for appositional bone growth between the borders of two bones. Increase in the size of the cranial vault takes place via primary growth of bone at the sutures, which forces the bones of the vault away from each other. Growth of the midface takes place via intrinsically determined sutural expansion of the

circummaxillary suture system, which forces the midface downward and forward. Mandibular growth takes place via intrinsically determined growth of the cartilage of the mandibular condyle, which pushes the mandible downward and forward.

There is considerable amount of growth occurring in suture (Baer MJ, 1954, Enlow & Hunter 1964) and hence from this point of view sutural growth attains significance. But on the basis of definition of growth center by Baume, sutures cannot be called as growth centers.

Sicher postulated that bone growth within the various maxillary sutures produces pushing of the bone which results in forward and downward movement of maxilla. It was believed that the stimulus for bone growth is tension, produced by the displacement of bones. Koski (1968) stated that there are two different views regarding the structure of sutures. The first school of thought (Sicher and Weinman) considers sutures (Fig. 5.11A) as a three layered structure. It stated that the connective tissue between the two bones plays the same role as the cartilage at the base of the skull and like epiphysis of long bones. There is spreading of suture due to proliferation of middle layer of the sutural tissue. According to this concept, tissue separating force exists in the suture itself (Fig. 5.12A).

The second school of thought (Pritchard, Scott and Girgis, 1956) sees the suture as a five layer structure (Fig. 5.11B). Each bone at the suture has its own two layer periosteum on both sides and the intervening fifth layer between these periosteal layers. This layer plays a role in adjustment between the bones during growth, while the active proliferating role is played by the cambial layers of the periosteum of each bone. It is very clear now from the histological evidences that the sutural structure is not identical to that of the epiphyseal growth plate (Fig. 5.12B).

Sicher also perceived the mandible as a long bone and the mandibular condylar cartilage as comparable to epiphyseal plate.

Evidences against Sutural Theory

- Trabecular pattern in the bones at the suture change with age, indicating the changes in the direction of growth. It cannot be accepted that sutures will have the necessary information for altering growth.

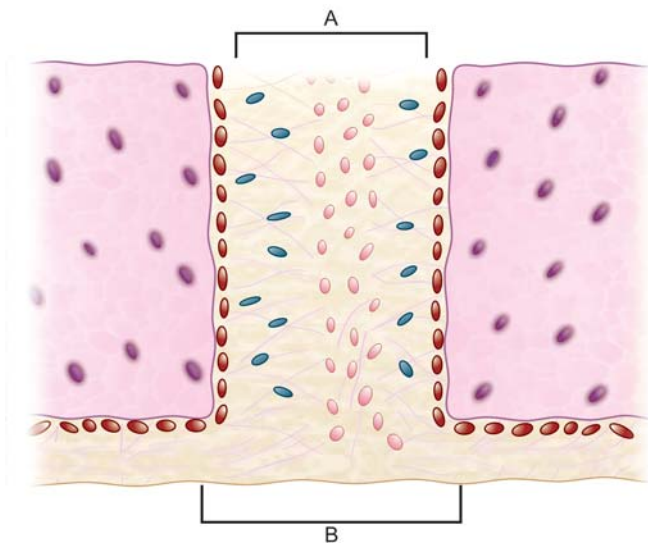


Fig. 5.11: A schematic illustration of the two differing views on the structure of the suture. 'A' represents the three-layer concept; 'B' the five-layer concept (*Source: AJO-DO 1968 566-583*): Cranial growth centers: Facts or fallacies?—Koski, 1968;566-83)

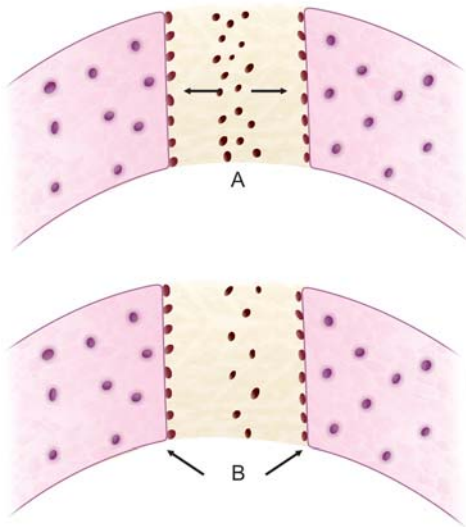


Fig. 5.12: A schematic illustration of the two differing views on the function of the suture. 'A' represents the concept of a thrusting force residing in the sutural tissue itself; 'B' represents the concept of an outside force separating the bones from each other. *Source: Koski AJO-DO 1968;566-83* Cranial growth centers: Facts or fallacies?

- Subcutaneous autotransplantation of the zygomaticomaxillary suture in the guinea pigs has not been found to grow (Watanabe M Laskin).
- Extirpation of facial sutures has no appreciable effect on the dimensional growth of the skeleton (Sarnat, 1963).
- Shape of sutures have been found to depend on functional stimulus (Moss & Salentejin, 1969).
- Closure of suture appears to be extrinsically determined (Moss ML).
- Sutural growth can be halted by mechanical force like clips placed across the sutures (Leitunen, 1956).
- The parallelism of circummaxillary suture so as to effect a forward and downward growth of maxilla is only superficial. Growth at zygomaticomaxillary suture occurs predominantly in lateral direction. The direction of growth of maxilla ranges from 0 to 82° in relation to SN plane (Fig. 5.13). It is practically impossible for the sutures running in the same direction to push the maxilla parallel to the reference plane (Bjork).

Conclusion

Present evidences indicate sutures as adaptive growth sites. Sutural tissues have no tissue separating force and they are not comparable to growth centers.

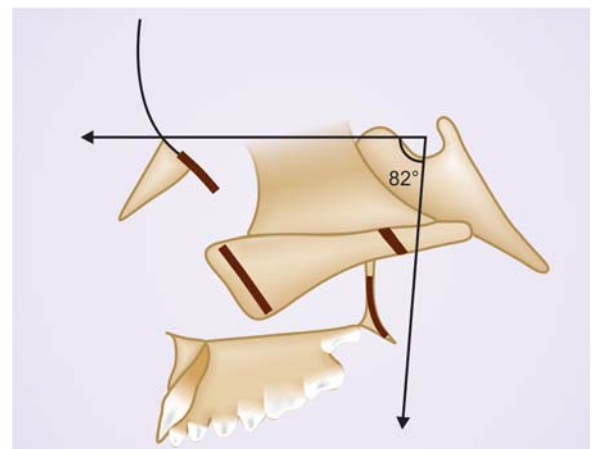


Fig. 5.13: A schematic illustration of the nearly parallel superficial direction of the main sutures of the upper face and of the range of direction of maxillary growth (After Björk. Acta Odont Scandinav 1966;24:109-127)

Scott Hypothesis/Nasal Septum Theory/ Cartilagenous Theory/Nasocapsular Theory

James H Scott, an Irish anatomist proposed the nasal septum theory as the single and unified theory of craniofacial growth.

Essence of Theory (Fig. 5.14)

According to the nasal septum theory, sutures play little or no direct role in the growth of the craniofacial skeleton. Sutures are considered as merely passive, secondary and compensatory sites of bone formation and growth. After recognizing the importance of cartilaginous parts of the head, nasal capsule, mandible and cranial base in prenatal growth, Scott felt that this cartilagenous development was under tight genetic control and was of the opinion that they continued to dominate postnatal facial growth also. Scott concluded that nasal septum is mostly active and vital for craniofacial growth both prenatally and postnatally. The anteroinferior growth of the nasal septal cartilage which is buttressed against the cranial base "pushes" the midface downward and forward (Figs 5.15 and 5.16).

The cranial base synchondroses cause the growth of the cranial base and Scott compared the condylar cartilage to the cranial base cartilage.

Discussion

Numerous experimental studies were conducted to address the validity of Scott's hypothesis. Latham elaborated Scott's ideas and also emphasized the role of septomaxillary ligament in the growth of midface beginning from the prenatal period till 3 or 4 years of life. He stated that maxillary suture begin as sliding joints and later manifest in increasing osteogenesis, contributing to the displacing force. He combined the ideas of Sicher and Scott. This theory is based on the fact that cartilage is a pressure adapted tissue and expansion of cartilage

provides the force to displace maxilla downward and forwards. According to Scott, bone separation must precede before the adaptive sutural bone growth occurs. The bone separation, he feels, is because of growth of organs like brain, eyeball or cartilage. Scott is of the opinion that there are two suture systems:

1. Posterior suture system lies behind the maxilla and separates it from palatine, lateral mass of ethmoid, lacrimal, zygomatic and vomer bones.

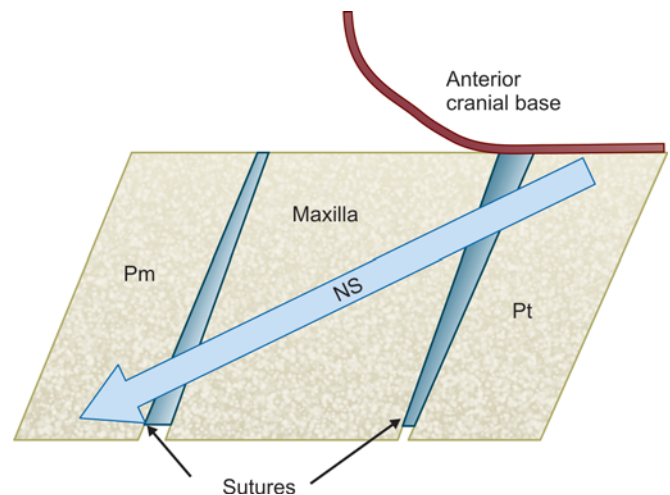
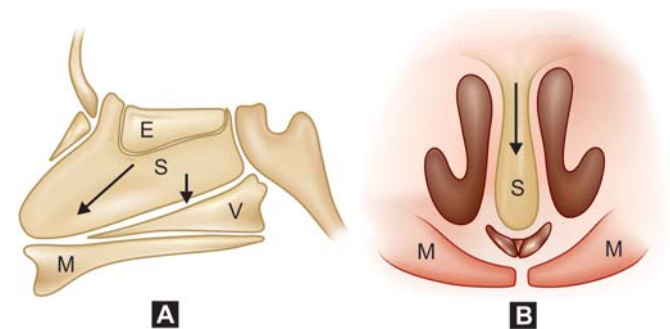


Fig. 5.15: Schematic representation of the nasal septum theory of craniofacial growth. Growth of the nasal septal cartilage pushes the midface downward and forward relative to the anterior cranial base. This results in a separation of the midfacial suture system, which then fills in via secondary, compensatory sutural bone growth (Source: Semin Orthod 2005;11:172-83)



Figs 5.16A and B: Schematic illustration of the cartilaginous nasal septum, of its relation to the neighboring structures, and of its alleged growth directions. (A) Sagittal view; (B) Frontal view; E, ethmoid bone; M, maxilla; S, septum; V, vomer. Source: Cranial growth centers: Facts or fallacies?—Koski AJO 1968; 566-83

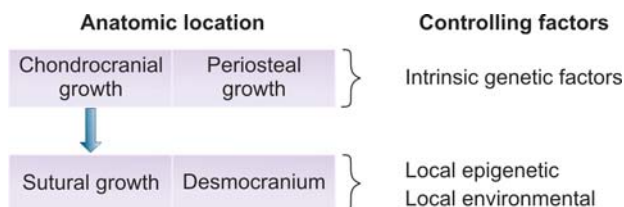


Fig. 5.14: Controlling factors of growth according to Scott

2. Anterior suture system separates premaxilla, nasal and vomer bone. The second suture system disappears in the human face during later part of fetal period and or after birth.

Scott said that nasal septal cartilage is an extension of the cranial base cartilage and as it grows further, it separates the facial bones from one another and also from the cranial portion of the skull. Thus it allows bone growth to take place at the sutures (frontomaxillary, frontonasal, frontozygomatic, and frontozygomaticomaxillary) by surface deposition.

Evidences Supporting the Theory

- Extirpation of septal cartilage in growing rats resulted in deficient growth of the snout (Sarnat, 1966).
- Latham and Burstone (1966) concluded that nasal septum has a role in determining anteroposterior growth of upper face.
- Burstone emphasized the importance of the septal growth impulse to maxillary growth in cleft palate cases. Failure of the underdeveloped maxillary segment to unite with nasal septum in complete unilateral clefts deprives it of the growth impulse or energy. The normal contralateral side on the other hand, attained normal growth.
- Sarnat and Long undertook auto radiographic studies with thymidine to determine levels of proliferative activity of cartilage cells. They found increased proliferative activity of the cells in the posterior regions of nasal septum which reflects endochondral ossification in this region.
- Sarnat in 1988, from experiments on rabbit snout concluded that deformity of snout after resection of nasal septum was the result of lack of growth.
- Steinler, Kvinslaw compared the increase in size of autotransplanted nasal septum in subcutaneous abdominal wall in rats. All the autotransplanted nasal septums showed increase in height and length and they retained the shape. This suggests that nasal septum has intrinsic growth potential.
- Latham (1974) described the role of septopremaxillary ligament passing from anteroinferior border of nasal septum to anterior nasal spine and intermaxillary suture in the premaxillary region. He stated that the traction through the ligament will exert downward and forward growth of maxilla.

- Koski after histological study of nasal septal cartilage found that there is endochondral ossification taking place at septoethmoidal junction.

Evidences Against the Theory

- Moss and Bloonberg (1968), Brigit Thilander (1970) found only slight deformity after extirpation of septal cartilage. They concluded that septal cartilage provides only mechanical support for the nasal bones and is not a primary growth center.
- Melson (1977) stated that downward sliding of vomer in relation to anterosuperior part of nasal septum takes place throughout craniofacial development making it unlikely that cartilaginous septum could push the maxillary complex forward as suggested by Scott.
- Moss stated that malformation in snout following excision of nasal septum is due to trauma following surgery.
- Burstone and Latham reported a case with missing nasal septum. The child had normal resorption and deposition of palate, height of upper face. Only sagittal development was affected.

Conclusion

At present, nasal septum theory is still accepted as a reasonable explanation for craniofacial growth. Nasal septum may be important for anteroposterior growth of face because of endochondral growth process occurring at its posterior border. It is not considered to be an active contributor for vertical development of face.

Functional Matrix Hypothesis (FMH)—Melvin Moss

- Introduction
- Essence of theory
- Definition
- Explanation
- Functional cranial analysis of maxilla
- Functional cranial analysis of mandible
- Neurotrophism
- Constraints of functional matrix hypothesis

Introduction

The concept that "form follows function" was first proposed by Vander Klaaw (1948-52). Functional

matrix theory is actually an extension of this concept. Melvin Moss and his co-workers developed the form and function concept into the "functional matrix hypothesis". Introduced in 1960s, Moss's view point became a principal catalyst of a new way of looking at craniofacial growth which became known as the functional paradigm.

Essence of the Theory

The basic principle of the functional matrix hypothesis is simple. Functional matrix hypothesis maintains that apart from initiating the process of development, heredity and genes play no active role in growth of skeletal structures in general and craniofacial skeleton in particular. The craniofacial skeleton develops initially, and later grows in direct response to the extrinsic epigenetic environment (Fig. 5.17). Moss states that *bones do not grow—bones are grown*. More precisely, the FMH claims that epigenetic, nonskeletal factors or process are the prior, proximate, extrinsic and primary cause of all adaptive, secondary responses of skeletal tissues and organs. The responses of the skeletal unit are not controlled by informational content of the intrinsic skeletal cell genome. But it is controlled by the functional matrix operations. Proponents of the functional matrix theory states that the expansion of the soft tissue matrix is primary and the bone growth is purely a secondary and compensatory event. Translation of the various bones of the face is due to volumetric expansion of the encapsulated spaces or tissues.

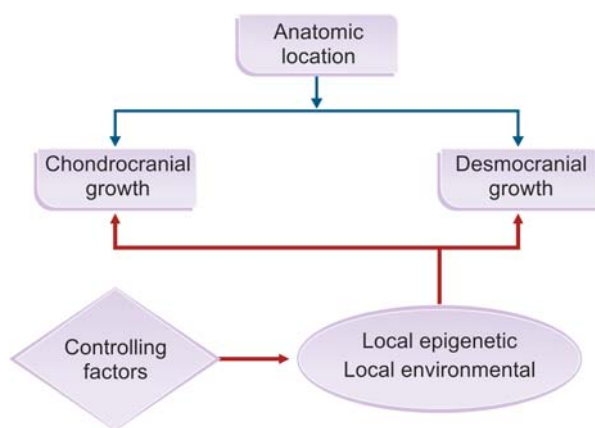


Fig. 5.17: Controlling influences on growth according to Moss

Definition

Functional matrix hypothesis claims that the origin, growth and maintenance of all skeletal tissues and organs are always secondary compensatory and obligatory responses to temporally and operationally prior events or processes that occur in specifically related nonskeletal tissues, organs or functioning spaces (functional matrices).

Explanation (Fig. 5.18)

To understand the factors that affect craniofacial bone growth. It is necessary to understand the local environmental and resultant skeletal structure in terms of their functional cranial component.

Functional Cranial Component

The functional matrix hypothesis considers the head, not the skull as the region of the body where a number of operations are carried out. So, head is a composite area of individual encapsulated areas within which specific functions like respiration, digestion, olfaction, vision, neural integration are performed. To perform each function, certain hard and soft tissues are required. The totality of all the skeletal structures, soft tissues and functioning spaces (nasal, oral, etc.) necessary to carry out a specific function is collectively called a "functional cranial component".

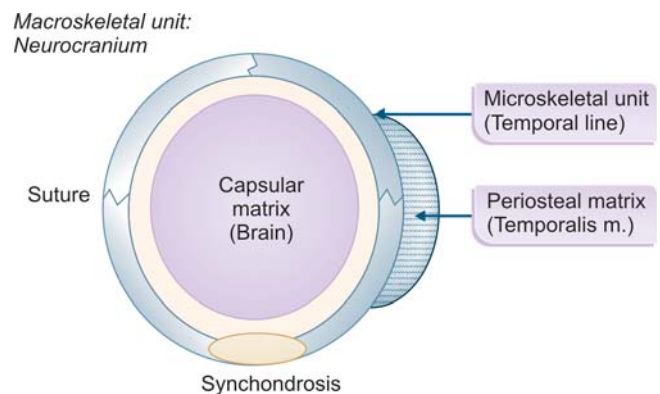


Fig. 5.18: Diagrammatic representation of functional matrix theory. Primary growth of the *capsular matrix* (brain) results in a stimulus for secondary growth of the sutures and synchondroses, leading to overall enlargement of the neurocranium (*macroskeletal unit*). Function of the temporalis muscle exerts pull on the *periosteal matrix* and bone growth of the temporal line (*microskeletal unit*) (Source: Semin Orthod 2005;11:172-83)

Each functional cranial component consists of a *skeletal unit* and a *functional matrix* (soft tissues and spaces). Any function is actually performed by the functional matrix, while the skeletal unit provides the necessary biomechanical role of providing protection and support to the soft tissue matrix. The skeletal unit may be composed of many bones, a single bone or a small portion of a bone. There are two types of skeletal units: micro-skeletal unit and macro-skeletal unit. The functional matrix consists of two distinct types: the *periosteal matrix* and the *capsular matrix*. The activity of both the matrices is essential for craniofacial growth.

Functional Matrix

The functional matrix refers to all the soft tissues and spaces that perform a given function.

- The *periosteal matrix* corresponds to the immediate local environment. They are virtually self defining. Examples of periosteal matrices include muscles, blood vessels, nerves, teeth etc. The effects of periosteal matrices are best exemplified by the effect of muscles upon the skeletal units. Lack of contraction leads to atrophy of the bone. All periosteal matrices act homogeneously by means of osseous deposition and resorption. The muscles are attached either into the skeletal tissue or indirectly by fusion with the outer fibrous layer of the periosteum. Functioning muscles influence developmental changes in the form of skeletal tissues to which they are attached. This is achieved through muscle bone interface (Moss, 1971). Sim and Kelly suggested that osseous blood flow adjusts to prior changes in osseous metabolism and they further noted that blood flow is increased at resorption site and depositary areas are poorly vascularised. The periosteal matrices stimulation causes growth of the micro-skeletal units. They act to alter the size or shape or both of the bones. The growth process that occurs due to periosteal matrix stimulation are called "transformation" (Fig. 5.20).
- The "capsular matrix" is defined as the organs and spaces that occupy a broader anatomical complex. The functional cranial components arise, grow and are maintained within a series of capsules. Each capsule is an envelope which contains a series of functional cranial component, skeletal units and their related functional matrices and is sandwiched between two covering layers. In the neurocranial capsule, this cover

consists of skin and duramater, while in the orofacial capsule the skin and mucosa form this limiting layer. All spaces intervening between functional components themselves and between them and the limits of the capsule are filled with indifferent loose connective tissue. Each capsule surrounds and protects a capsular functional matrix. The neurocranial capsular matrix consists of the brain, leptomeninges and CSF (Fig. 5.19). It is easy to visualize the neurocranial capsule. On the other hand, orofacial capsular matrices or functioning spaces are difficult to visualize. The capsular matrices exist as volume.

Neurocranial capsule: In the neurocranium, it is the volume of the total neural mass which is morphogenetically significant. The expansion of the enclosed and protected capsular matrix volume is the primary event in the expansion of the neurocranial capsule. As the capsule enlarges, the whole of the included and enclosed functional components, that is the periosteal matrices and the micro-skeletal units are carried outward in a totally passive manner. The calvarial functional cranial components as a whole are passively and secondarily translated in space.

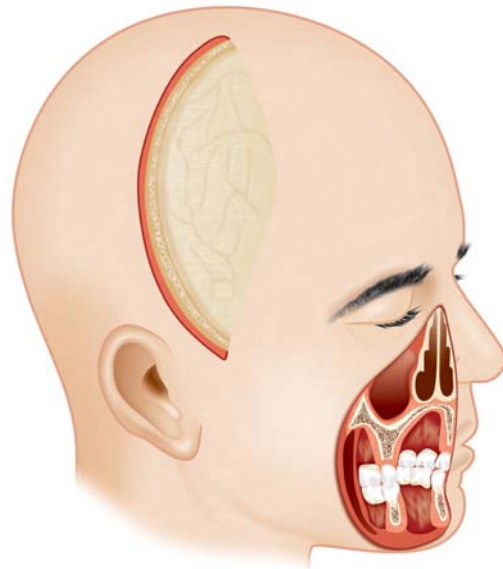


Fig. 5.19: The neurocranial and orofacial capsular matrices are shown. The neural capsular matrix consists of the entire neural mass, including the dura mater, while the orofacial capsular matrix consists of these functioning spaces. In both cases, the skeletal units exist completely within their respective capsules. (Source: Moss and Salentijn. AJO 1969;20-31): The primary role of functional matrices in facial growth

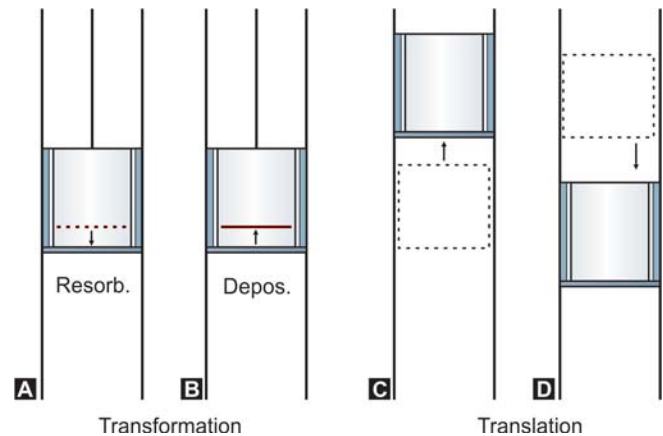
In experimentally induced or pathologic states, the periosteal matrices are prevented from exerting their morphogenetic activity. In the neurocranium, hydrocephaly is such a condition. The expansion of the neurocranial capsule is always proportional to the increase in neural mass. But in hydrocephaly, increase in intracranial pressure, obliterates vascular flow within the capsule and so prevents periosteal accretion of bone at sutural areas, thus producing the characteristic large fontanelles, and other sutural dehiscence. The point is simple. The neural skull does not grow first and provide space for the secondary expansion of the neural mass. Rather, the expansion of the neural mass is the primary event which causes the secondary and compensating growth of the neural skull.

Orofacial capsular matrix: The orofacial capsular matrix or oropharyngeal functioning spaces is surrounded by the orofacial capsule. Limiting layers of this cavity are skin on the external aspect and mucous membrane internally. Establishment of the morphogenetic primacy of the orofacial functioning spaces will cause translation of all skeletal units embedded within the orofacial capsule. The human oronasopharyngeal space increases in size from the third month of pregnancy. This volumetric increase produces a compensatory increase in the size of the orofacial capsule. Growth of the capsule results due to mitosis of both the epithelial and mesenchymal cellular elements and the resultant increase in intercellular materials which cause an expansion of the capsule. As the capsule enlarges, both the periosteal matrices along with the respective skeletal units are passively and secondarily translated to a new position in space.

Thus the enclosed capsular matrices act indirectly on the macroskeletal units or on entire functional cranial component. They do not act by the process of osseous deposition or resorption or by affecting cartilages directly. They do not alter the size or shape of the skeletal units; instead they change their location in space. This type of growth process is called "translation" (Figs 5.20A to D).

Skeletal Unit

The skeletal unit refers to the bony structures that support the functional matrix and these are necessary or permissive for that function. The skeletal unit does



Figs 5.20A to D: Diagrammatic representation of transformation (A, B) and translation (C, D). Note in transformation there is change in size or shape whereas in translation there is change in position

not refer to the individual bone directly, but to the function it supports. There are two types of skeletal units:

1. Microskeletal,
2. Macroskeletal units.

Microskeletal units are parts of the bone whose growth is modulated by the periosteal matrices. Functional variations in the periosteal matrices may be expressed within the microskeletal unit (Fig. 5.21). The possible interaction between periosteal matrix and microskeletal unit includes—temporalis-coronoid process, masseter, medial pterygoid-gonial angle, teeth-alveolar bone. The change in size and shape of microskeletal units occur independently of the changes in spatial position. Moss uses two terms for this: "transformation" or "intraosseous growth".

Macroskeletal units are made of the core of maxilla, mandible and neurocranium. Moss and Greenberg pointed out that the basic maxillary unit is the core which supports and protects the infraorbital neurovascular triad and in mandible, the basal tubular portion which protects the mandibular canal. Through the neurotrophic influence, the spatial constancy of the infraorbital canal with respect to anterior cranial base and mandibular canal from foramen ovale through mandibular foramen to mental foramen is maintained (*Unloaded nerve concept*). The capsular matrix expansion causes the macroskeletal unit to passively change the position. This process is called translational growth of skeletal structures.

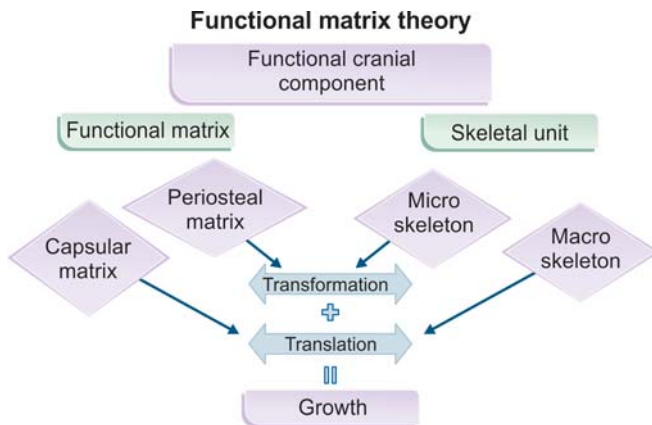


Fig. 5.21: Organization of functional matrix theory

The overall skeletal growth is a combination of changes in microskeletal and macroskeletal units due to stimulation of periosteal and capsular matrices respectively. This total growth change is termed "interosseous growth" by Moss.

Functional Cranial Analysis of Maxilla (Fig. 5.22)

From a functional point of view, there is no such entity as maxilla. Rather, we have a bone composed of several relatively independent, functional skeletal units that is associated with many functions which includes vision, respiration, digestion, speech and protection of neurovascular structure.

The *basal bone* designates the maxillary skeletal unit which serves to protect and support the infraorbital neurovascular triad. It is that portion of the maxilla that is left over when all the other maxillary skeletal units have been subtracted. Moss and Greenberg point out that the basic functional matrix for the basic skeletal unit is the infraorbital neurovascular triad.

Of the three components of this triad, it is the maxillary division of the trigeminal nerve that plays the major role in maintaining the spatial constancy of the infraorbital canal to the anterior cranial base. Thus, it indirectly produces a similar constancy of the spatial position of the basal maxillary skeletal unit relative to the anterior cranial base.

The area of infraorbital foramen is the site of the first ossification of human maxillary bone. (Premaxilla, being a separate entity). Bone formation begins at about the

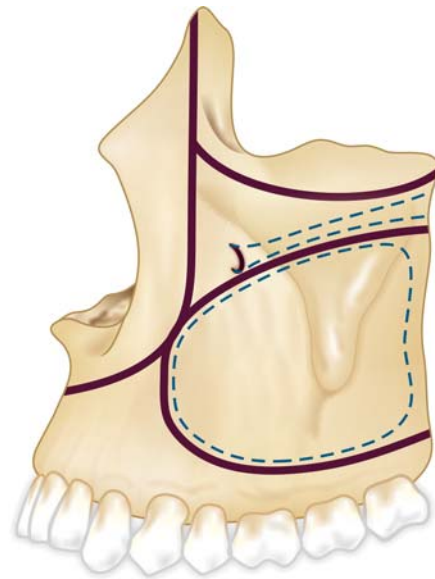


Fig. 5.22: Functional cranial analysis of maxilla in lateral view (Source: Angle Orthodontist: Functional cranial analysis of human maxillary bone: Melvin Moss and Greenberg, July 1967)

end of sixth week in the form of radiating trabeculae. They get transformed into a smooth bordered plate like bone. At all the ages, the horizontal position of the curved infraorbital canal as viewed in Norma verticalis is lateral to the maxillary dentition with only minor exception.

The orbital mass functional matrix virtually ceases their volumetric growth by the end of first decade. The definitive height of the nasal cavity is attained at the same time. Mostly all the functional matrices that might affect the position of maxillary basal skeletal unit come to rest at this time and do not participate in further growth of the maxillary complex.

The nonbasal maxillary matrices related to oral and dental function continue to grow after 10 years of age.

The facial bones are enclosed within an orofacial or splanchnocranial capsule. So the maxillary base is passively carried downwards, forwards and laterally as a result of expansion of their capsule (orbital, nasal, oral matrices). In the maxilla, as the several related orofacial matrices expand, the maxilla would tend to be carried away from adjacent bones. This causes adaptive "bone fill in response". The growth seen in the orbital floor is an example for this secondary and compensatory growth event following the passive movement of maxilla. In the anteroposterior direction, the forward passive motion

of maxilla is constantly being compensated by bone deposition posterior to the maxillary tuberosity and to the palatal processes of both the maxillary and palatine bones.

Moss and Greenberg further state that there are three types of bone growth changes seen in maxilla. Firstly, these are associated with compensation for the passive motion of the bone brought about by the primary expansion of the orofacial capsule. These changes help to maintain anatomical and functional continuity between maxilla and adjacent bones. Secondly, there are changes in bone morphology associated with alterations in absolute volume, size, shape or spatial position of any or all of the several relatively independent maxillary functional matrices like orbital mass. Finally, there are bone changes associated with the maintenance of the form of the bone itself. The posterior repositioning of the zygomatic arch which accompanies relative forward movement of the arch is an example for this.

It must be noted that all three processes do not take place simultaneously. But the concept of differential or sequential expression of differing growth phase has been emphasized by people like Walker.

Moss found a relatively constant position of infraorbital foramen both in anteroposterior and vertical plane.

Functional Cranial Analysis of Mandible

Mandibular structure is meaningful in terms of its function. The mandibular matrix consists of:

- All muscles with mandibular attachments
- Neurovascular triads (arteries, veins and nerves)
- Associated salivary glands
- The teeth
- Fat, skin and connective tissues
- The tongue
- The oral and pharyngeal spaces.

Mandible is situated and it grows and functions within the matrix. Moss speaks of mandible as a group of micro-skeletal units and a basal core part (Fig. 5.23).

Thus, the coronoid process is one microskeletal unit under the influence of temporalis muscle: gonial angle is another microskeletal unit under the influence of masseter and pterygoid muscles. The alveolar base is the microskeletal unit for teeth. The basal tubular portion serves as a protection for the mandibular canal and it follows a logarithmic spiral in its downward and forward

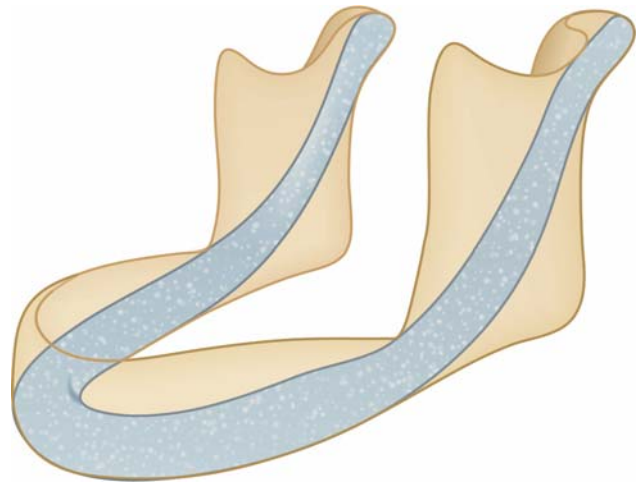


Fig. 5.23: Mandible: Core and microskeletal units. Stippled part denotes the core of mandible

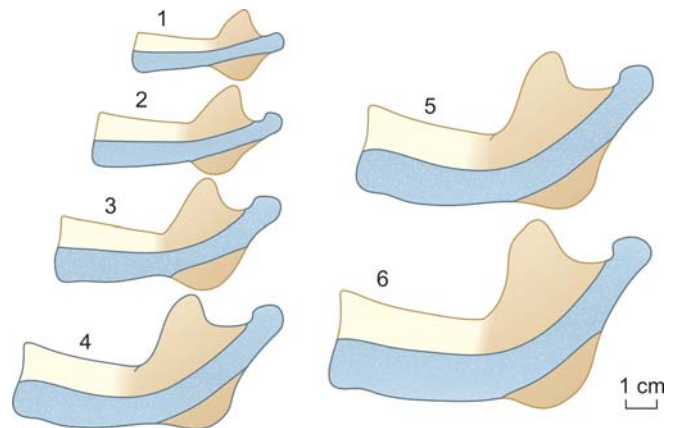


Fig. 5.24: Protected nerve concept. The central core is straight first. Later it follows the logarithmic curve

movement from beneath the cranium. This is called "unloaded nerve concept". The most constant portion of the mandible is the arc from foramen ovale to the mandibular foramen and mental foramen (Fig. 5.24).

Mandibular growth demonstrates the integrated activity of periosteal and capsular matrices in facial growth. Orofacial capsular matrix growth causes an expansion of the capsule as a whole. The enclosed and embedded, microskeletal unit is passively and secondarily translated to successively new positions. Evidences point out that the growth of the functional mandibular matrix is probably the primary event which causes the relocation of the mandible in space and the increments of condylar

length and microskeletal unit growth are secondary and compensatory events.

Important concepts in mandibular growth: According to Moss, three important phenomena occur during mandibular growth:

- *Constancy of the relative position of mental foramen in the mandibular corpus:* If the horizontal body is divided into premental and postmental segments and these segments when measured at different ages, it was found that the length of these two segments remains relatively proportional throughout life (Fig. 5.25). This proves the point that increase in corpus length cannot be solely due to condylar growth as this would increase the relative size of the postmental segment.
- *Absolute migration of the dentition through the alveolar bone:* This movement which is different from mesial drift happens during the first two decades. While the position of the mental foramen remains constant, the relationship of mandibular dentition to it does not. This migration is most pronounced during the eruption of permanent dentition.
- *Change in the direction of mental foramen:* Mental foramen can be compared to the nutrient foramen of long bones. When a pin is placed in such a foramen, the protruding head of the pin, "points to the more rapidly growing end". Figure 5.26 shows that in newborn, such a pin in mental foramen points forward while its direction is upward at 6 years and relatively backward in the adult. The reason for this is given by LaCroix. It is claimed that the periosteum of growing bone is under tension and that the tensile force at a given point is proportional to the growth rates of the two ends of the bone. When the growth rate of one end predominates, the periosteal tension in that direction will be greater. The effect of such unequal tension is "slipping" of the periosteum and consequent migration of the point of entry of the nutrient vessel (Fig. 5.27). This along with surface apposition of new bone which accompanies growth in width causes the foramen to face in the direction of most rapid growth.

In the newborn, formation of chin is the most rapid mandibular growth process and therefore foramen faces forward. With the eruption of permanent teeth, the increase in corpus height due to alveolar growth causes

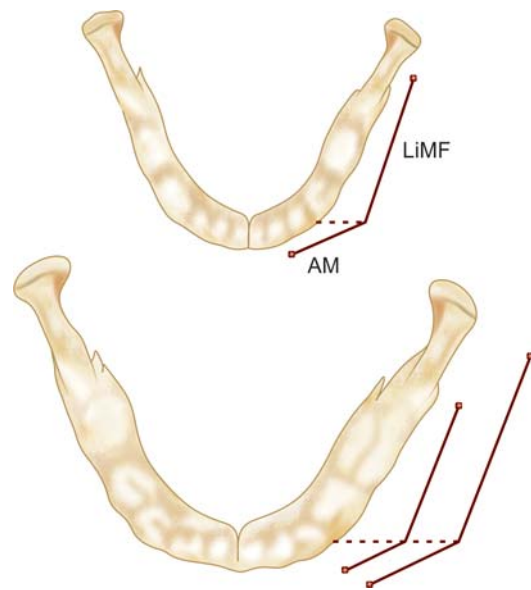


Fig. 5.25: Comparison of fetal and newborn mandibles. Dimensional increase in the premental (AM) and postmental (LiMF) foraminal segments are constantly proportional

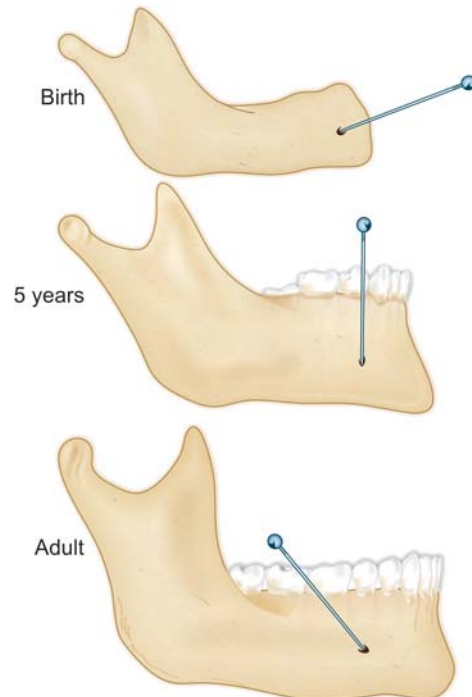


Fig. 5.26: Newborn, 5 years old and adult mandibles. Note the variations in the direction of head of the pin

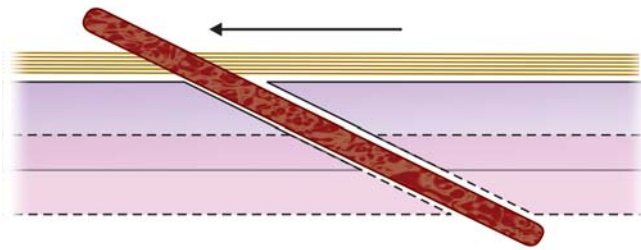


Fig. 5.27: Sliding of the periosteum indicated by the arrow causes displacement of the nutrient canal. Continuous lines show the canal in its present form. Dotted lines show the canal at an earlier stage. Arrow denotes the active growth site (After LaCroix)

the foramen to face upward. Subsequent addition to corpus length and posterior shift of the ramus which occurs with eruption of permanent 2nd and 3rd molars direct the foramen backwards.

These additional features of mandibular growth present a more dynamic concept. Mandible is not simply growing. It is not only adding to its length and height but also passively carrying the teeth downward and forward. Other events like shift in greatest growth rate, migration of entire dentition mesially and maintenance of constant position of mental foramen with alteration in direction of opening also take place.

Transmission of Functional Stimulus to the Bone, Neurotrophism

Most important aspect of FMH is the mechanism by which the functional response or stimulus is converted or translated to the skeletal interface and the way they are regulated or controlled. Moss says it is through a process called neurotrophism. Neurotrophism is defined as a *non-impulsive transmissive neurofunction involving axoplasmic transport providing for the long-term interaction between neurons and innervated tissue, which homeostatically regulate the morphological, compositional and functional integrity of those tissues.*

Singer, 1963 explained neurotrophic function as fundamental expressions of neurocellular activity "indicating that the "nervous system" is also concerned with the integrity of body structure". Gutman states that these functions act homeostatically to maintain and renew the structures and functional capacity of body parts.

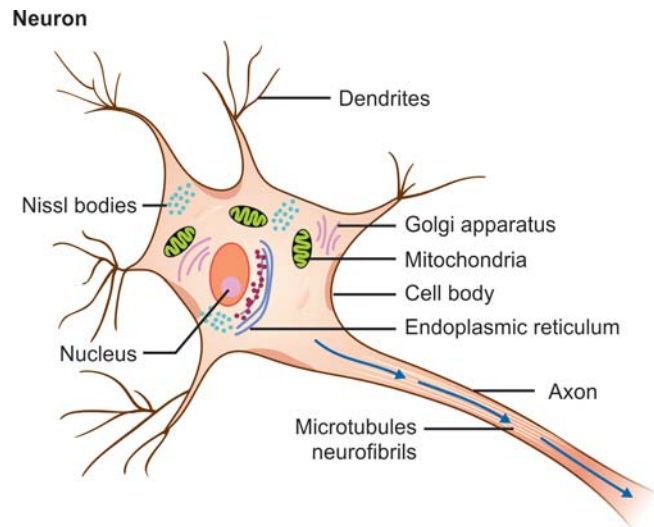


Fig. 5.28: Schema of presumed pathway of neurotrophic substances denoted by blue arrows

Types of neurotrophism: Moss classifies neurotrophism into three types arbitrarily:

1. Neuromuscular
2. Neuroepithelial
3. Neurovisceral

Neurotrophism unlike other nervous functions deals with non-impulsive conductive function of neurons. This requires an additional cellular process by which the trophic functions are carried out. "Axoplasmic streaming" or transport is the term used to describe this function of nonimpulsive conduction of neurons (Fig. 5.28).

Neuromuscular trophism: Embryonic myogenesis is not under the control of nerves and neurotrophism. Neural innervations are established at the myoblast stage of differentiation. Moss states that after this stage, skeletal muscle ontogenesis cannot proceed without innervations. Diculescu et al wrote that the complex chain of events leading to particular expression of the genetic embryonic potential is not fully present within the cell and it also includes information from the nerves. Samaha, Guth and Abers found that new species of proteins has been synthesized and suggested that nerve influences genetic expression of the cells. Accordingly Moss stated that genetic control cannot reside solely in the functional matrices alone and there is neurotrophically regulated homeostatic control of the genome. He also believes

that similar neurotrophic mechanism exists for capsular matrix which passively regulate the functional cranial component.

Muscle denervation—reinnervation: Muscle denervation and subsequent reinnervation enable us to differentiate effect on muscle tissue associated with the loss of impulse conduction and muscle contraction from those due to loss of neurotrophic factor. If motor neurons are sectioned and the related muscles subsequently become reinnervated, there is reformation of muscle tissue and it grows even before the recovery of neuronal conductive function. This demonstrates neuromuscular trophism.

Cross innervations: Experimental cross innervations procedure wherein the first nerve is cut and the free ends are placed in muscles supplied by slow nerves and vice versa were carried out. After a recovery period, it was seen that fast muscles became slow and slow muscles became fast (Previtt and Safesky). This change is brought about by neural influence which has a direct effect on the contraction.

Hyperneuralization: Hyperneuralization refers to the ability of the muscle fiber to have more than one motor end plate. When the usual nerve, innervating a muscle is crushed, the muscle responds to experimentally implanted second motor nerve by the formation of the new end plate. The original end plate gets reestablished after the original nerve recovers. It is important to note that an already innervated muscle fiber normally will not form end plate with an implanted second nerve. The following points can be concluded from the study:

- Neurotrophism effects do not depend upon the presence of end plate.
- Neurotrophic effects could be produced by a motor nerve, while the same nerve is totally incapable of eliciting a contractile response from the same muscle.
- Neurotrophic material is diffusible and does not require end plate apparatus (Lentz).

Neurovisceral trophism: In the orofacial region, salivary gland is partially trophically regulated. Increase or decrease of mature salivary gland, under neurotrophic influence have been experimentally demonstrated.

Neuroepithelial Trophism

The neurological work of neurotrophism first began in the field of dermatology. Examples include areas of

sensory loss, skin lacks its ability to withstand trauma: trophic ulcers are first caused by normal protective capability of the skin and second by lack of sensory function. The factors which contribute to neuroepithelial trophism are:

- Local mechanism operating in areas of high mitotic activity.
- Epithelial growth factors.
- Type of feedback mechanism between dermis and epidermis.
 - Amphibians' limb regeneration is an example for neuroepithelial trophism. It happens only after intimate neuroepithelial contact.
 - Presence of taste buds is dependent upon intact neural innervations (Joseph 69).
 - The taste buds undergo degeneration along with thinning of adjacent epithelium following denervation. These examples make it viable to think, whether the functioning oral pharyngeal spaces which are lined by epithelium are trophically regulated.

Neurotrophic control of genetic activity: Neurotrophic control of genetic activity is demonstrated in many tissues under experimental conditions:

- Protein synthesis in oral epidermal cells and specific enzymatic synthesis in taste buds epithelium appear to be neurotrophically regulated (Guth 63, Robbin 70).
- Handlman and Wills suggest control of salivary glands through autonomous nerve fibers.
- It is suggested that the regenerating nerve exert a direct control on the synthesis of DNA, RNA and protein in regenerating tissues (Thornton 70).
- Leborvitz and Singer 70 found that the neural tissue homeogenicity possess the ability to regulate protein synthesis.
- It is also suggested that nerve influences gene expression in the muscle cell.

Even though FMH gained popularity, it suffered from a major drawback. Moss was not able to clearly explain the process by which the functional stimuli could get converted into a signal and affect changes in bone. In his series of articles titled functional matrix hypothesis revisited, Moss tries to explain the FMH in a more detailed and at microscopic level and validate FMH.

Constraints of Functional Matrix Hypothesis

Initially, the FMH provided only qualitative description of the biologic dynamics of cephalic growth at gross anatomic level. The two explanatory constraints of FMH are:

1. Methodological
2. Hierarchical

Methodological Constraint

FMH used only macroscopic measurement by using point mechanics and arbitrary reference frames like cephalometric radiograph. This permitted only method specific description that cannot be structurally detailed. This constraint was overcome by using continuum mechanics techniques of the finite element method and of the related macro and boundary element methods. This method added quantitative aspects of localized cephalic growth kinematics to the earlier qualitative description of growth dynamics.

Hierarchical Constraint

The second constraint of FMH is it does not explain how the extrinsic, epigenetic functional matrix stimuli are transduced into regulatory signals at the cellular, multicellular or molecular levels. The prior explanations were suspended between these two hierarchical levels, i.e. the cellular and multicellular or tissue level. The epigenetic or sum of all lower attributes (biophysical, biochemical, genomic) could not explain or predict the higher attribute of the bone tissue.

The new version of FMH tries to bridge the gap between hierarchical constraints and explains the operation from genome to organ level by two concepts:

1. Mechanotransduction occurring in single cells.
2. That bone cells function multicellularly as a connected cellular network.

Mechanotransduction

Mechanotransduction is the process by which a mechanical stimulus is converted into a biologic signal to affect a cellular response. Whenever there is alteration in the external environment, the vital cells are perturbed. Mechano-sensing enables a cell to sense and to respond to the external stimuli by using *mechanoreception*. After the signal is received, it is transferred to intracellular signal by *mechanotransduction*. The process of

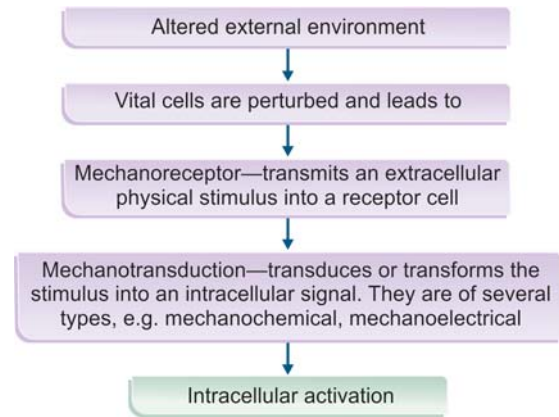


Fig. 5.29: Process of mechanotransduction

mechanotransduction is explained in Figure 5.29. Bone adaptation requires intracellular transmission of the transduced signals.

Osseous Mechanotransduction

Bone is subjected to constant loading, both static and dynamic. This is essential for normal homeostasis of bone. When the threshold value of the force is exceeded, the loaded tissue responds to the stimulus by the triad of bone cell adaptation. The triad includes bone deposition and maintenance and bone resorption. Both osteoblasts and osteocytes are competent for intracellular stimulus reception and transduction.

The osseous mechanotransduction has four unique properties:

1. Bone cells are not cytologically specialized like other mechanosensory cells.
2. Single bone loading stimulus evokes three adaptational responses, whereas nonosseous process generally evoke one.
3. Osseous signal transmission is aneural; it does not involve neural pathways unlike other mechanosensory signals.
4. The adaptational response is confined within the individual bone.

Osseous mechanotransduction translates the periosteal functional stimulus into a skeletal unit cell signal by two skeletal cellular mechanotransductive processes: "ionic" and "mechanical". The ionic or electrical processes involve some form of ionic transport through the bone cell plasma membrane. The possible ionic process

includes *stretch activated ion channels, electro-mechanical, electrokinetic and electric field strengths*. The basis for mechanical process is the physical conductivity of the transmembrane molecule integrin. This molecule is connected extracellularly with the macromolecular collagen of the organic matrix and intracellularly with cytoskeletal actins. Actins, in turn are connected to the nuclear membrane where the mechanical action induces a series of intranuclear processes.

Thus by this interconnected physical chain of molecular levels, the periosteal functional matrix may regulate the genomic activity of the skeletal unit bone cells.

Role of Osseous Connected Cellular Network (CCN)

After the initial mechanotransduction, wherein the external stimulus is converted into intracellular mechanosensation, Moss tries to explain the intercellular transmission of the signal through the connected cellular network hypothesis.

Bone as an Osseous Connected Cellular Network

All the bones are extensively interconnected by gap junction and form an osseous connective cellular network. Osteoclasts are an exception to this. Gap junctions are found where the plasma membranes of a pair of canalicular processes meet. Canaliculi form extensive communication between osteons and interstitial regions. Gap junctions also connect superficial osteocytes to periosteal and endosteal osteoblasts. All osteoblasts are also interconnected laterally. Vertically they connect periosteal osteoblasts with preosteoblastic cells and this in turn is interconnected. Thus each CCN is like a true syncytium and are electrically active.

It is said that mechanotransductively activated bone cells like osteocytes can initiate membrane potential which gets transmitted through CCN. Gap junctions which allow bidirectional flow of information are the cytological basis for the oscillatory behavior of CCN. Moss has outlined the following features of CCN:

- Developmentally, skeletal CCN is an untrained, self organized, self adapting and epigenetically regulated system.
- Operationally it is a stable, dynamic system that exhibits oscillatory behavior permitting feedback.

- Structurally, an osseous CCN is non modular, i.e. the variations in its organization permit discrete processing of differential signals. It is this property that permits the triad of histological responses following a signal loading event.

Moss also states the osseous CCN is different from messenger activation. Thus the functional stimuli after intercellular activation goes hierarchically upward again through histological levels to the event of gross bone form adaptational changes (Fig. 5.30).

Moss in an effort to further validate the functional matrix theory presented the two articles titled: genomic thesis, epigenetic antithesis and the resolving synthesis. The definitions for various mechanisms given by Moss are explained in Box 5.1.

Conclusion

Moss concluded by saying that both genomic and epigenetic factors are necessary and sufficient causes individually. Together both provide the necessary and sufficient cause for the control of morphogenesis. But epigenetic process and events are the immediate proximate cause of development and as such they are the primary agencies.

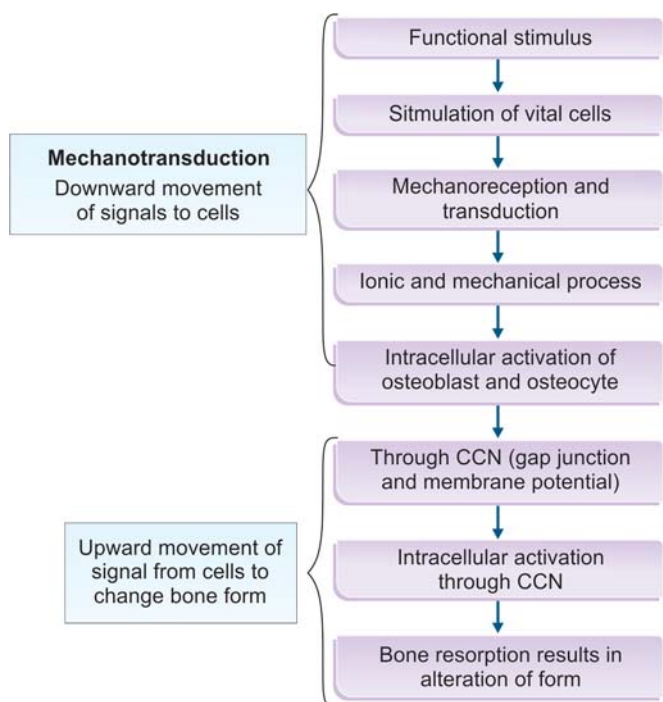


Fig. 5.30: Hierarchical conversions of functional stimuli

Box 5.1: Biological mechanisms and process defined by Moss

Epigenetics: It is defined as the sum of all of the extrinsic factors impinging on vital structure including mechanical loading and electroelectric states and all of the intrinsic biophysical, biomechanical, biochemical and bioelectric microenvironmental events occurring on, in, and between individual cells, extracellular materials, and cells and extracellular substances.

Hierarchy: Biologic structures are hierarchically organized with structural and functional complexity increasing "upward" from, the level of subatomic particles to protons, electrons, atoms, molecules, subcellular organelles and on to cells, tissues, organs organisms.

Emergence: Emergence consists of the appearance, at each successively higher and structurally and/or operationally more complex level of new attributes or properties not present in the lower levels, whose existence or function could not in any way be predicted, even from a complete knowledge of all the attributes and properties of any or all of the preceding lower organization level.

Causation: Is concerned with how the attributes of a given biologic structural level "cause" (control, regulate, determine) the attributes of the next higher level.

They may be categorized as either intrinsic (material & formal) and extrinsic (efficient). Material & formal cause are classified as "prior" causes which mean existing before the creation of some specific state or structure. They are intrinsic because they reside within the vital structure intra and intercellularly. Efficient cause is proximate, i.e. its operation immediately causes the creation of a new state. Efficient causes are extrinsic and they represent the epigenetic mechanisms.

Process: A process is a series of activities or operations that lead towards a particular result.

Mechanism: A mechanism is the fundamental physical or chemical process involved in or responsible for an action-reaction or the natural phenomenon. A mechanism stimulates a process.

Von Limborgh's Compromise Theory (Box 5.2)

Von Limborgh after review of the sutural theory, cartilaginous theory and functional matrix theory has summarized the following features:

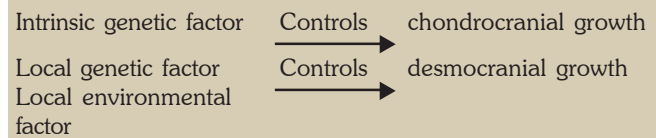
- Intrinsic genetic factor controls chondrocranial growth.
- Epigenetic factors originating from skull cartilages and head tissues control desmocranial growth.
- Local environmental factors like tension forces and pressure influences the growth of desmocranial growth.
- General epigenetic and general environmental factors are less significant in craniofacial growth.

Modern Composite Theory (Fig. 5.31)

The composite theory tries to explain the growth of maxilla and mandible. It separates the facial skeleton into desmocranium, chondrocranium and splanchnocranium. Calvarium forms the desmocranium, the cranial base and nasal septum as chondrocranium. Remaining part of middle face and mandible constitute the splanchnocranium.

Essence of the Theory

Chondrocranium is considered the dominant factor in craniofacial growth. Postnatal remnants, cranial basal

Box 5.2: Essence of Von Limborgh's theory

cartilages (sphenooccipital synchondroses predominantly) and the nasal cartilages act as growth centers. These are influenced by intrinsic genetic factors. As shown by the blue arrow in the Figure 5.31 S-O synchondroses exert a direct action on the desmocranium. Local epigenetic (capsule) and local environmental (periosteal matrix) then control the calvarium. Sutures are considered only as growth sites. Another cartilage mechanism responsible for craniofacial growth is nasal cartilage. The growth of nasal cartilage pushes the maxilla downward and forward.

The growth of mandible seems to be controlled by both local epigenetic and local periosteal factors. The position of mandible is also affected by cranial base flexion and growth by altering the posture of glenoid fossa (denoted by red arrow in Figure 5.31). Ventral position of maxilla with respect to glenoid fossa also affects the mandible by rotating it forward or backward. Therefore

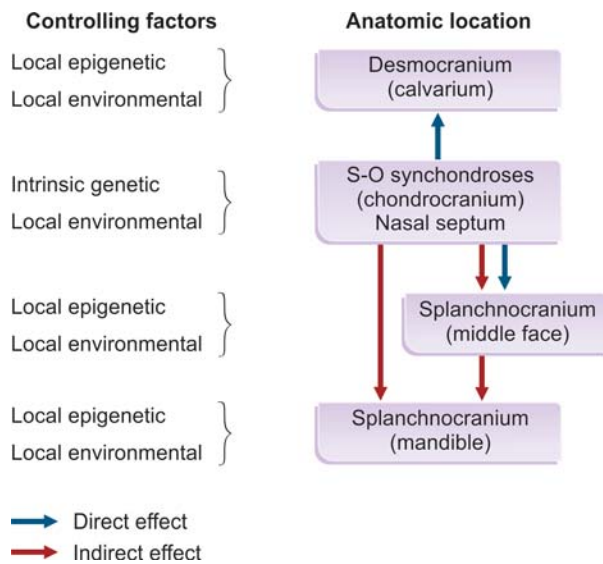


Fig. 5.31: Craniofacial growth as explained by composite theory

the position of mandible is affected by the bones in closed proximity to it.

Servo System Theory: Alexandre Petrovic

Petrovic, using the language of cybernetics explained that the growth of various craniofacial regions is the result of interaction of a series of causal changes and feedback mechanisms. Based on a series of experiments, Petrovic and co-workers have formulated a cybernetic model for the control of mandibular growth.

Essence of the Theory

According to the servo system theory, control of primary cartilages (mid face) takes a cybernetic form of "command" whereas control of secondary cartilages like condyle is comprised of both direct effect of cell multiplication and also indirect effects. Simply stated, the servo system theory is characterized by the following two principal factors: (1) The hormonally regulated growth of the midface and anterior cranial base, which provides a constantly changing reference input via the occlusion, and (2) The rate-limiting effect of this midfacial growth on the growth of the mandible. While growth of the mandibular condyle and of the sutures may be affected directly and indirectly by systemic hormones, growth of these structures is clearly more compensatory and

adaptive to the action of extrinsic factors, including local function as well as the growth of other areas of the craniofacial complex.

Cybernetics

Servo system theory starts with the explanation of cybernetics. Weiner defines cybernetics as the science of control and communication in the animal and machine.

Ashby defines it as the study of systems that are open to energy but closed to information and control. Cybernetics theory postulates that every thing affects everything and therefore organized living systems never operate in an open loop manner. Open loop is a type of feedback mechanism. The other type of feedback is closed loop mechanism. The feedback closes the regulation loop of a given system in the following way.

Input → Measure of effect → Return of modified information → Regulation of effect.

According to cybernetic theory, the behaving organism is not seen as a passive respondent called into action by the changing environmental stimuli but as a dynamic system which continuously generates intrinsic activity for organized interaction with the environment.

Cybernetics in Craniofacial Growth

Cybernetics demonstrates the relationship between observational and experimental findings. It is a tool for better understanding of clinical problems and complex nature of craniofacial morphogenesis. The hierarchical relationship of servo system is shown in Figure 5.32.

Black box: The physiologic system under investigation is represented by the black box. The contents of the black box is usually not known.

Feedback signal: It is the function of controlled variable that is compared to the reference input. It is negative in regulator and servo system.

Closed loop system: If a physiologic system is designed to maintain a specific correspondence between inputs and outputs, in spite of disturbances, it is called as closed loop system. It is characterized by the presence of a feedback loop and comparator. Closed loop has two variations namely regulator and servo system.

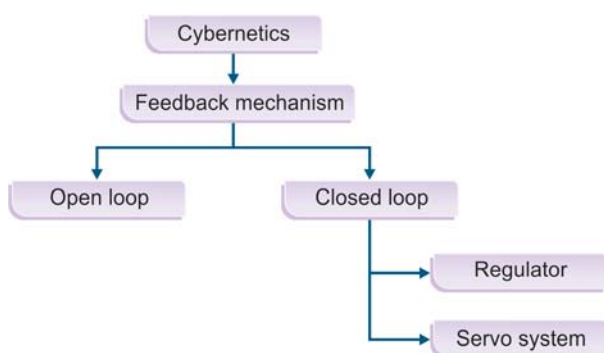


Fig. 5.32: Approach to servo system concept

Open loop system has no feedback loop or comparator.

The regulator: The main input is a constant feature in this system. The comparator detects disturbances and their effects. It is a negative feedback system: disturbances cause changes that tend to restore the normal state of the disturbed system to the initial state.

The servo system: It is also called as follow-up system. The main input is not a constant in this system but varies across in time.

Elements of Servo System Theory (Fig. 5.33)

- **Command** is a signal established independently of the feedback system under scrutiny. It affects the behavior of the controlled system without being affected by the consequences of this behavior. *Examples:* Secretion rates of growth hormone, testosterone, estrogen, and somatomedin. They are not modulated by variations of craniofacial growth.
- **Reference input elements:** Establish the relationship between the command and reference input. Includes septal cartilage, septo-premaxillary ligament, labionarary muscles, premaxillary and maxillary bones.
- **Reference input** is the signal established as a standard of comparison, e.g. sagittal position of maxilla. Ideally it should be independent of the feedback.
- The **controller** is located between the deviation signal and the actuating signal.
- The confrontation between the position of the upper and lower dental arch is the **comparator** of the servo system.

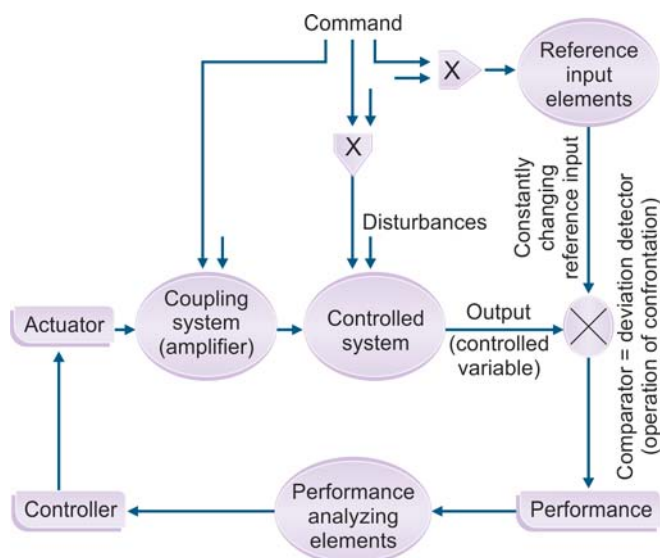


Fig. 5.33: Elements and organization of servo system theory

- Activity of the retrodiscal pad and lateral pterygoid constitutes the *Actuating signal*. The elastic menisco-temporal and menisco-mandibular frenum of the condylar disc form the retrodiscal pad.
- **Controlled system** is between the actuator and controlled variable, i.e. is growth of condylar cartilage through the retrodiscal pad stimulation.
- **Controlled variable** is the output signal of the servo system. Best example is sagittal position of mandible
- **The gain:** The gain of a system is the output divided by input. Gain value greater than one is called amplification and if it is less than one it is called attenuation. The pterygocondylar coupling is an example for gain.
- **The disturbance:** Any input other than the reference required is called a disturbance. Disturbance results in deviation of the output signal. For example, increase in hormone secretion results in supplemental lengthening of mandible.
- **The attractor:** This is the final structurally stable state in a dynamic system. It includes the full cusp class I molar relation.
- **The repeller:** This includes all unstable equilibrium states like cusp to cusp occlusal relationships.

Explanation of the Theory (Fig. 5.34)

According to servo system theory, the midface grows downward and forward under the primary influence of

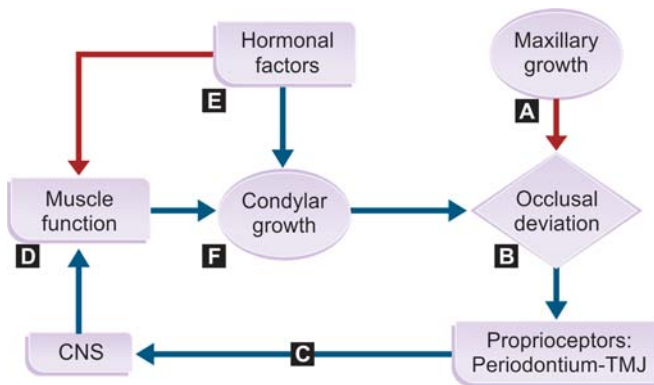


Fig. 5.34: Servo system theory of craniofacial growth, with emphasis on the growth of the mandible. Anterior growth of the midface (A) Results in a slight occlusal deviation between the maxillary and mandibular dentitions (B) Perception of this occlusal deviation by proprioceptors (C) Triggers the protruder muscles of the mandible to become more active tonically (D) In order to reposition the mandible anteriorly. The muscle activity and the protrusion in the presence of appropriate hormonal factors (E) Stimulate growth at the mandibular condyle (F). (Source: After David Carlson. *Semin Orthod* 2005;11:172-83)

the cartilaginous cranial base and nasal septum, influenced principally by the intrinsic cell-tissue related properties common to all primary cartilages and mediated by the endocrine system. The influence of somatotrophic hormone on the growth of cartilages of nasal septum, sphenoccipital synchondroses and other synchondroses follows that of a cybernetic form of command pattern. Related to this event, the maxillary dental arch is carried into a slightly more anterior position. This is the first and primary event.

This causes a minute discrepancy between the upper and lower dental arches, which Petrovic referred to as the "comparator", that is, the constantly changing reference point between the positions of the upper and lower jaws. Upper dental arch is the constantly changing reference input. Second, proprioceptors within the periodontal regions and temporomandibular joint perceive even a very small occlusal discrepancy and tonically activate the muscles responsible for mandibular protrusion. Petrovic says the functional appliances will work in the same way when given to stimulate mandibular growth in class II malocclusions (Fig. 5.35).

Third, activation of jaw protruding muscles (Retrodiscal pads and lateral pterygoid muscles) acts

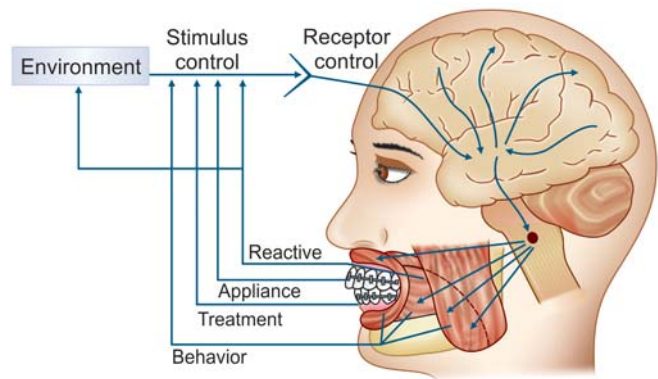


Fig. 5.35: Feedback displacements which cause condylar growth stimulation

directly on the cartilage of the mandibular condyle and indirectly through the vascular supply to the temporo-mandibular joint, stimulating the condyle to grow. The growth in secondary cartilages like condyle corresponds to local and environmental factors (Epigenetic control). Lower arch constitutes the controlled variable.

Finally, the effect of the muscle function and responsiveness of the condylar cartilage is influenced both directly and indirectly by hormonal factors acting principally on the condylar cartilage and on the musculature.

This entire cycle is continuously activated as a servomotor as long as the midface-upper dental arch continues to grow and mature and appropriate extrinsic, hormonal, and functional factors remain supportive. This affects the output signal. The output signal is the final sagittal position of mandible. The sagittal position of mandible depends on the modification of condylar growth by the activity of retrodiscal pad and lateral pterygoid muscle stimulation.

Evidences Against the Theory

- Goret-Nicaise, Awn (1983), found that the resection of the lateral pterygoid muscle fails to diminish condylar growth.
- Whetten and Johnston (1985) used a bilateral condylotomy model in young rats to test the extent to which direct muscle traction can alter the rate of condylar growth and removed the lateral pterygoid muscle unilaterally. They found no difference in condylar growth between the two sides.

- Das, Myer and Sicher (1980) found that the occlusion remained unaffected in condylectomy studies.

Conclusion

This theory is similar to condylar epiphysis hypothesis in terms of importance attributed to growth of condylar cartilage. It differs in that Petrovic says that condylar growth can be modified therapeutically or in response to functional requirements. The major strength of the servo system theory is that it provides a road map for future research and experimentation.

Rate Limiting Ratchet Hypothesis (Johnson)

The rate limiting ratchet hypothesis views the condyle as an opportunist. Unable to grow when loaded but able to grow when unloaded. It is suggested that the condyle is in effect a functional rectifier, a ratchet whose growth is the ultimate determinant of downward and forward mandibular translation.

The hypothesis is based on the finding that condyles have an inherent ability to grow and pressure will arrest their growth. Yozwiak (1979) demonstrated that condyles do not grow in the face of pressure. According to the ratchet hypothesis, condylar growth and the resultant mandibular growth is conditional upon the unloading of the condyle. Functional loading controls the mandibular growth while functional unloading stimulates the mandibular growth. Johnston (1986) suggests therefore that the pattern of condylar loading is the only signal necessary to control condylar growth. Mandibular distraction unloads the condyle and thereby permits it to exercise its intrinsic ability to grow. This growth thus serves to preserve a fraction of the amount of mandibular distraction that unloaded the condyle and results in a downward and forward mandibular translation. The condyle is therefore considered as a "rate limiting ratchet". A given amount of condylar growth would in turn make permanent, a like amount of the mandibular functional displacement and thus would constitute a necessary and rate limiting precondition for normal translatory growth.

Therefore, according to this theory, anything or any therapeutic appliance that increases the amount of time a condyle is unloaded would be expected to increase the condylar growth and ultimately the length of the condyle. Conversely any appliance that increases the

amount of time condyle is loaded would be expected to decrease the condylar growth and thereby results in a shorter mandible.

Growth Relativity Hypothesis (John C Voudouris 2000)

Growth relativity refers to growth that is relative to its displaced condyles from actively relocating fossae. John C Voudouris introduced this concept to explain the possible effect of functional appliances on condyle and the resulting growth.

The main foundations of growth relativity hypothesis are:

- Displacement of condyle
- Nonmuscular viscoelastic tissue stretch
- Force transduction beneath the fibrocartilage of the glenoid fossa and condyle add new bone formation

Displacement of Condyle

The displacement that takes place initially following mandibular advancement affects the fibrocartilagenous lining in the glenoid fossa to induce bone formation locally.

Viscoelastic Stretch

Once the condyle is displaced, it is followed by the stretch of nonmuscular viscoelastic tissues. Viscoelasticity refers to all the non-calcified tissues (Fig. 5.36). Viscoelasticity addresses the viscosity and flow of the synovial fluids, the elasticity of the retrodiscal tissues, the fibrous capsule and other nonmuscular tissues including LPM, perimysium, TMJ tendons and ligaments, other soft tissues and bodily fluids. Due to viscoelastic stretch there is influx of nutrients and other biodynamic factors into the region, through engorged blood vessels of the stretched retrodiscal tissue that feed into the fibrocartilage of the condyle. Alteration of synovial fluid dynamic also takes place.

Force Transduction and New Bone Formation

This is the most interesting aspect wherein new bone formation takes place at some distance from actual retrodiscal tissue attachments in the fossa. The glenoid fossa and the displaced condyle are both influenced by the articular disk, fibrous capsule and synovium which

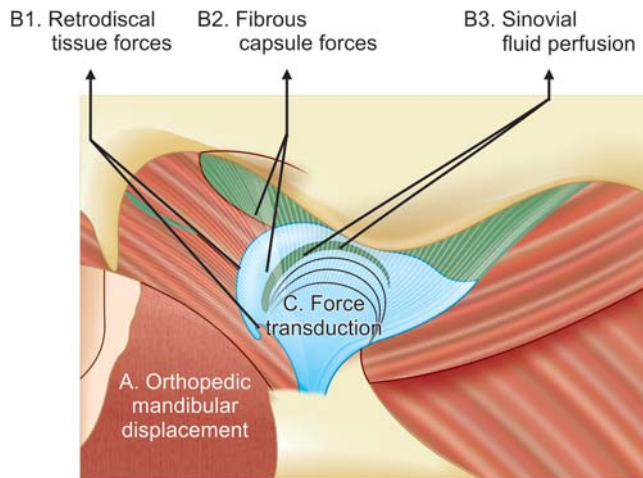


Fig. 5.36: Growth relativity hypothesis for condylar and glenoid fossa growth with continuous orthopedic displacement. Three factors influence growth modification: A, displacement; B, viscoelastic tissue pull (arrows); and C, transduction with fibrocartilage. Viscoelastic tissues include B1, superior and inferior bands of the retrodiscal fibers; B2, fibrous capsule (fine white lines); and B3, synovial fluid perfusion in a posterior direction. The pull of the retrodiscal fibers, capsule, and the flow of synovial fluids on the condyle relative to the glenoid fossa are in a posterosuperior direction. The forces are translated to the condyle with the articular disk's posterior, anterior, lateral and medial (collateral) attachments. (Source: Am J Orthod Dentofacial Orthop 2000;117:247-66)

are contiguous. Thus condylar growth is affected by viscoelastic tissue forces through attachment of the fibrocartilage that covers the head of the condyle.

Effect of three growth stimuli (Displacement + viscoelasticity + transduction of force): Modification of growth occurs by a combination of all the three factors: Modification "first" occurs as a result of the action of anterior mandibular displacement. "Second", the condyle is affected by the posterior viscoelastic tissues anchored between the glenoid fossa and "third", displacement and viscoelasticity further stimulate the normal condylar growth by transduction of forces over the fibrocartilage cap of the condylar head. Voudoris and Kuftinec compares this process to the light bulb analogy (Fig. 5.37). The resultant increase in new bone formation appears to radiate as multidirectional finger like processes beneath the condylar fibrocartilage and significant appositional bone formation is seen in the fossa. Growth relativity hypothesis is more specific to condyle only when compared to functional matrix hypothesis.

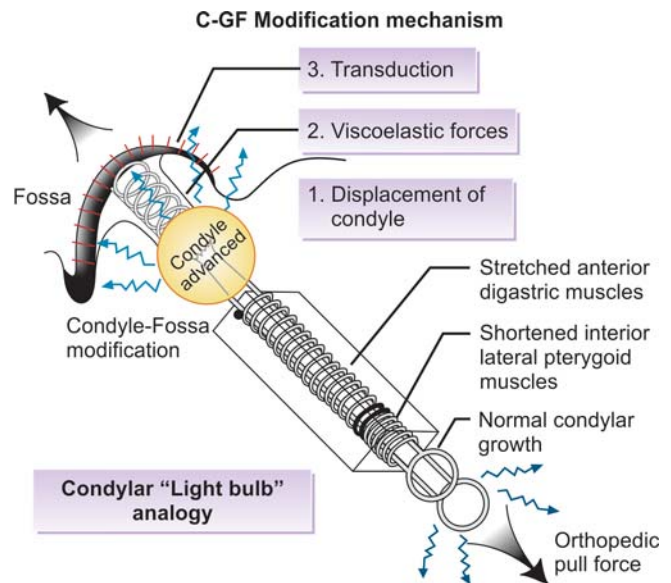


Fig. 5.37: Light bulb analogy of condylar growth and retention. When the growing condyle is continuously advanced, it lights up like a light bulb on a dimmer switch. When the condyle is released from the anterior displacement, the reactivated muscle activity dims the light bulb and returns it close to normal growth activity. In the boxed area, the upper open coil shows the potential of the anterior digastric muscle and other perimandibular connective tissues to reactivate and return the condyle back into the fossa once the advancement is released. The lower coil in the box represents the shortened inferior LPM. The open coil above the yellow condylar light bulb represents the effects of the stretched retrodiscal tissues. (Source: Am J Orthod Dentofacial Orthop 2000;117:247-66)

Conclusion

Identifying the primary trigger mechanism for the growth of maxilla and mandible will help the orthodontist to either stimulate or retard the growth of maxilla and mandible. This will prove to be the key to successful growth modification treatment in skeletal malocclusions.

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Chapter

6

Postnatal Growth of the Craniofacial Skeleton

CHAPTER OUTLINE

- Postnatal Growth of Cranial Vault/Calvaria
- Cranial Base
- Nasomaxillary Complex
 - Maxilla
 - Palate
 - Zygomatic bone
 - Nasal cavity
 - Orbit
- Mandible
- Temporomandibular Joint
- Dynamics of Facial Growth

Face or the countenance is the window of man's mind to the world. Human facial skeleton is unique; craniofacium is formed of 22 bones, 8 cranial and 14 facial bones inclusive of the mandible, the movable bone of face. Study of the postnatal growth of the craniofacial skeleton is intriguing because the remodeling pattern of the face is highly programmed, selective and specific. An infant's face is almost round, the length and width of the face are equal and cranium : face ratio is about 1:8. The face appears to be tucked under the cranial bones at birth, and with growth literally grows out of the cranium. As age advances, the increase in length of face is more than the other dimensions (width and depth) such that in average individuals, the facial height is twice as much as it is in the immediate postnatal period. The remodeling pattern is highly specific; structures like zygomatic bone, chin, superciliary arches become prominent with age. Apposition/resorption pattern functions towards attainment of the adult facial forms. Growth of face is not just specific but also differential and follows a pattern. Cephalocaudal gradient of growth is also seen in the face, there is an increasing axis of

growth as a person ages; cranial vault completes growth earlier than the base. Similarly, maxilla completes early, but mandible though completing growth late, has better potential for growth modification.

A phylogenetic view at the facial form will reveal a reptilian face with a flat upper face and a long protruding lower face with a snout. Requirements for survival have led to the enlargement of the frontal (forebrain) and the temporal lobes of the brain. The massive enlargement of brain in man is associated with flexure of the cranial base to accommodate the growing brain. Increase in size of the frontal lobe is responsible for the unique feature in man, the forehead. Growth of the temporal lobe causes redirection of orbit from lateral to the front, and subsequently, the two prominent facial bones, the maxilla and mandible come to be redirected downward. The long snout of the reptilian face is lost to a flatter mid and lower face of man with the new forehead, in man.

Research of postnatal growth of face has been made systematic by studying the face regionally. Craniofacial skeleton can be divided into cranial and facial skeleton. Cranium, in turn can be divided into cranial vault and cranial base while the facial skeleton can be studied under the nasomaxillary complex, mandible, and the temporomandibular joint.

POSTNATAL GROWTH OF CRANIAL VAULT/CALVARIA

Cranial vault or desmocranium is formed of 8 bones (2 parietal; 1 occipital; 1 frontal; 2 sphenoid (part of the greater wing); 2 temporal). The ossification of bones of desmocranium is intramembranous in nature. At birth,

the cranial vault is 63 percent of their adult size, the sutures are wide open, the cortex is thin and the area of contact of bones has only a fibrous covering, an extension of the periosteum. They are called fontanelles. There are six fontanelles in all, *anterior*: at the junction of frontal and parietal bones, *posterior*: at the junction of occipital and parietal bones, *posterolateral*: at the junction of temporal, occipital and parietal bones, *anterolateral*: at the junction of frontal, parietal, temporal and greater wing of sphenoid bones. The closure (ossification of the membrane) of fontanelles differs: posterior at birth, anterior at about first year, anterolateral around 15 months and posterolateral around 1½ years of age. The fontanelles are nature's way of assuring a smooth exit of the relatively larger fetal head out of the vaginal canal. Fontanelles can be palpated at infancy. Fontanelles also facilitate the postnatal growth of the brain. Neural tissue grows most during the first eight years of life after which there is hardly any growth. Synostosis or fused sutures and absence of fontanelles impede the growth of brain and studies have shown that surgical splitting of synostotic suture in certain syndromes encourages the growth of brain. As the brain grows, the bones of the cranial vault are passively translated in space with sutural split. To maintain contact with adjacent bones, osteogenic tissue fills in at sutural ends, remodeling occurs by apposition/ resorption.

The cortex is thin and inner and outer bony plates of desmocranium are close to each other and parallel at birth. With growth and gradual remodeling, the thickness of the bony vault increases; there is formation of frontal sinus, increase in width between the plates, and formation of diploe between the tables. The inner/ endocranial surface of cranial vault is more under the influence of the growing brain than the outer table which is more mechanically influenced by functional stress that leads to pneumatization of the skull.

Sutures secondarily help in the growth of desmocranium. The cranial vault increases in length by growth of cranial base with active response at the sutures, especially coronal and lambdoid sutures. The increase in width is attributed to fill-in ossification at the interparietal, parietosphenoidal, parietotemporal, etc. The sutural ends are initially relatively smooth but, with time, as the sutures fuse they become interdigitated. About 90 percent of growth is complete by 5 to 8 years of age but midsagittal or interparietal suture remain patent till the third decade of life. Increase in height is

due to growth of parietal suture at its articulation with occipital, temporal and sphenoid bones.

According to Wagemans (AJO 1988), the suture refers to "the entire complex of cellular and fibrous tissues lying between and surrounding the opposing edges of two skull bones and including the bony edges". Stages of development of sutures were given by Pritchard, Scott and Girgis. The stages are:

- Stage of approaching of the bony edges.
- Stage of meeting of the bony edges.
- Early growing stage.
- Late growing stage.
- Adult stage.

Weinmann and Sicher hypothesized that sutures are three-layered; two peripheral layers of dense connective tissue near the bone and a layer of cells in between (Refer Fig. 5.11). Pritchard, Scott and Girgis countered by claiming that sutures are five layered:

1. Two cambial layers near the bony edges (cellular).
2. Two fibrous capsular layer.
3. Highly vascular middle zone.

The first two layers are contiguous with the periosteum of the bone. The cell population mainly consists of osteocytic and fibrocytic cells. On a macroscopic level, suture can have edge to edge contact (butt end) or may be overlapping. End to end or flat plane sutures are found in the sagittal suture system of the skull. Overlapping sutures are also found in the skull. Sutures are extremely adaptive, the shear and compressive stresses even may lead to formation of secondary cartilage in the suture. The adaptive changes taking place in sutures to applied mechanical stresses were summarized by Linge. According to Linge, forces applied to sutures are first translated into deformation (mechanical) and later into cellular activity. According to him, the external mechanical forces are the primary forces. When they are applied to the craniofacial skeleton, they are absorbed and a series of secondary forces are produced. These secondary forces trigger a cascade of mechanical reactions like tissue deformation, displacement, etc. The mechanical reaction leads to a biological response. This transduction of forces into cellular activity is explained by piezoelectric effect. Distortion of the cell membrane leads to alteration in cAMP levels in the cells which is associated with change in the deposition/resorption rate.

Sutural growth is a secondary and adaptive mechanism to the growth of the bones. Sutures are growth sites. The growth of neural tissue is almost

complete by 8 years, thus the sutures of cranial vault cease to have active fill-in of bone after 15 years. Cortical remodeling, increase in the dimension (pneumatisation) of the sinuses, apposition in the anterior aspect of frontal bone, are some of the growth activities seen after 15 years. It is clear that the cranial vault follows the growth of brain, it approximates the neural curve in Scammon's growth curve; the cranial vault completes its maximal growth quite early in life after which a plateau is achieved. Premature fusion of sutures is seen in craniostenosis where the growing brain applies pressure causing bulging out of eyeball, and extreme intracranial pressure.

Increase in size of the desmocranium is synchronous with the growth of the brain. Brain is not in direct contact with the endocranial surface of the vault. It is enclosed inside the meninges; similarly the bones of the vault are also enclosed by the osteogenic membranes. Growth of the neural tissue leads to the passive displacement of the bones of calvaria outward. All the bones are translated in space and enclosed in their capsular matrices, the neurocranial capsule (the periosteum, leptomeninges, brain according to Moss). One should remember that it is not the active growth (primary displacement) of the bones itself; instead it is the secondary displacement of the bones due to expansion of capsular matrix that causes growth of the desmocranium. The bones of the calvaria are embedded in the capsule. Translation leads to splitting of the sutures which creates tension at the sutural edges. The ends of these sutures receive new bone deposits. Osteoblasts line the sutural edges and deposition of bone to approximate sutural ends and maintain the contact of adjacent bone commences. Thus, sutural growth is not the primary motive force for bone growth, in other words, sutures are not growth centers but active growth sites. Baume defines *growth site* as "the region of periosteal or sutural bone formation and modeling resorption, adaptive to environmental influences". Going by the definition, it is clear that sutures are indeed growth sites, experimental studies have also proven beyond doubt that sutural tissue when transplanted subcutaneously fail to grow independently. They do not have the intrinsic genetic potential for growth to be growth centers. Koski defines *growth centers* as places of endochondral ossification with tissue separation force, contributing to the increase in skeletal mass. This definition implies that the tissue separation force caused by proliferation of cells is the intrinsic genetic potential.

Sutures thus have only a secondary and adaptive type of growth. Sutures are highly responsive to external environmental influences. The sutural ends of the flat cranial bones are relatively smooth at birth. With age and remodeling changes, they become serrated. In some instances, spicules of bone may be formed between two bones bound by the sutures, these are called wormian bones. Deposition is not only seen at sutural ends but also in the region of fontanelles.

The calvarial bones do not just grow by fill-in ossification at the sutures but also by remodeling. The endocranial and ectocranial surfaces undergo apposition as the calvarium expands with the growing brain (Fig. 6.1). The endosteal surface undergoes resorption, initially the cortex is thin with the inner and outer tables approximating each other and parallel. Resorption of the endosteal surface is responsible for the formation of diploe within the calvarial bones. The idea of expanding brain causing growth of cranial bones may be mistaken to cause resorption of the endocranial surface of the calvaria but it has been proven that the endocranial and ectocranial surfaces receive deposition of bone. It is only the endosteal surfaces of the inner and outer tables that undergo resorption. The thickness of the cortex increases in the process with increase in the medullary space as well. The sutural ends are depository that causes increase in the whole size of the individual bone. As the remodeling progresses, curvature of the bone reduces and the bones become flatter (Fig. 6.2). Similarly, the anterior surface of frontal bone in the region of supraorbital rim undergoes bone apposition. This process accentuates the superciliary arches. The endosteal surface of the frontal bone undergoes resorption which expands the frontal sinus. The basic difference in the morphology of the endosteal surface of the vault and base has been attributed to be responsible for the vault being depository and base being resorptive. The cranial base is formed of endocranial

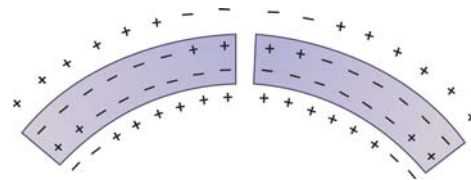


Fig. 6.1: Remodeling in the cranial vault. Notice the apposition in the ecto and endocranial surfaces. Resorption takes place in the endosteal surface, and formation of diploe

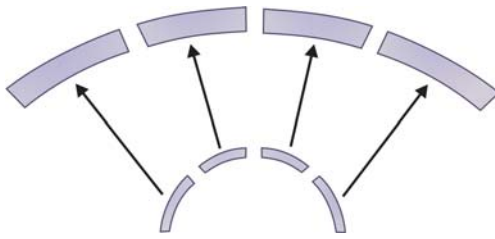


Fig. 6.2: Growth and expansion of vault cause flattening of bones

fossae which house the cerebral lobes thus bearing their weight. Growth at the sutures is not uniform throughout the cranial vault. Growth at the periphery (outer zone) near the calvaria is much more than the growth at the core of the brain stem. Also, sutural growth is more at the height of the calvaria and gradually decreases as the sutures approach the cranial base region.

CRANIAL BASE

Cranial base or floor is formed by endochondral ossification. In the prenatal life, cranial base is a large irregular piece of cartilage. The endocranial surface of cranial base is not flat, unlike the vault; it is divided into anterior, middle and posterior cranial fossae by bony elevations. The anterior cranial fossa is at a higher level than the middle cranial fossa which in turn is at a higher level than the posterior cranial fossa. The functional difference between cranial base and vault is vastly different, common function is the protection of brain. The cranial base: (i) Lodges all the lobes of cerebrum; (ii) Bears the weight of the rapidly expanding brain; (iii) Provides a passage way for all the cranial nerves exiting and blood vessels entering the brain; (iv) provides a thrust for the anterior growth of the facial skeleton. The alignment of cranial base itself is unique in humans. It is not flat and not in a plane as it is for other primates. Bipedal stance of man is associated with cranial base flexure. The middle and posterior cranial fossa are angulated, the whole cranial base appears to have flexed at the clivus. The angulation of clivus is around 65° . This flexure of cranial base has paved way for the redirection of face from forward to downward direction. The flexure is also responsible for the anterior and inferior movement of the middle face.

All the three fossae are clearly delineated by their own boundaries. The anterior and middle cranial fossae

by the lesser wing of sphenoid; the middle and posterior cranial fossae by the petrous temporal; the left and right anterior fossae by the midline bony ridge; the right and left middle cranial fossae by the sphenoidal body; the right and left posterior cranial fossae by clivus, foramen magnum, midventral bony ridge; and the right and left olfactory fossae are separated by the crista galli. All the fossae are resorptive and the elevations are depository. The cranial base is also a seat of a number of synchondroses. The numerous sutures contribute to adaptive bone growth. It is easy to understand that sutural remodeling will not be enough to compensate for the massively expanding lobes. Sutures are known to succumb under pressure and the weight of the lobes bearing down on the cranial base applies pressure to the sutures. Hence, cranial base growth may be attributed to the following causes, namely: (i) Displacement of bone due to expanding lobes of brain and growth at synchondroses; (ii) Secondary fill-in ossification of the sutures (playing a minor role); and (iii) Cortical remodeling. It is clear at one look that all the synchondroses are concentrated at the midline axis of the cranial base; hence elongation at the midline alone is contributed by synchondrosis. The orientation of cartilage cells of the spheno-occipital synchondrosis is also an evidence that only anteroposterior growth in the midline of cranial base is contributed by synchondrosis. The lateral expansion is mostly due to the expansion of lobes of the brain.

The anterior cranial fossa is formed by orbital plates of frontal bone and crista galli. Growth of forebrain in the initial stages of growth leads to anterior secondary displacement of the frontal bone (anterior wall of anterior cranial fossa). There is resorption in the floor of anterior cranial fossa to accommodate the growing brain and compensatory deposition at the roof of the orbit (Fig. 6.3). This displacement of bone displaces the entire bone inclusive of the outer and inner tables. The apposition and resorption pattern is just the reverse of that in prenatal life. In the fetal period, there is rapid growth of eyeball with the frontal lobe lagging behind. Deposition occurs in the cranial floor and resorption in the orbital roof. After birth, the frontal lobe catches up and surpasses the orbital matrix, thus reversing the cortical remodeling. The orbit develops like an expanding 'V' with deposition on the inside and resorption on the outside of the 'V'. After eight years of age, growth of

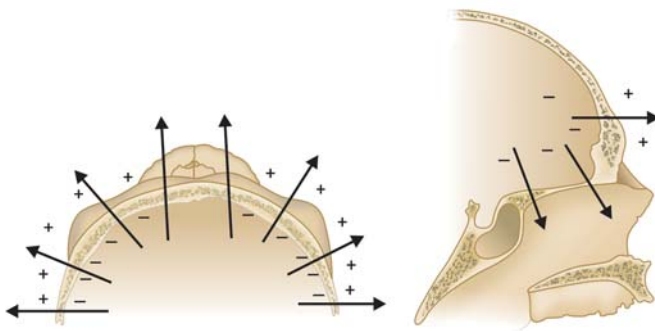


Fig. 6.3: Expansion of frontal lobe causes anterior growth of frontal bone. Resorption is seen in the fossa, deposition in the ectocranial surface

forebrain ceases and what follows thereafter is cortical remodeling. There is resorption in the endosteal surface of the outer table with deposition on the periosteal side. Deposition accentuates the superciliary arches. Frontal sinus also develops in the process. Displacement of bone splits and creates tension in the sutures leading to fill-in ossification. The sutures are frontotemporal, sphenofrontal, etc. The lengthening of anterior cranial fossa is directly due to growth of functional matrix. The area housing olfactory lobes (near the anterior terminus on either side of midline) are resorptive and the central midline ridge is depository (crista galli). The ectocranial surface of the cranial base is depository. Growth is not just in the anterior direction but also laterally and downward.

Temporal lobe is lodged in the middle cranial fossa on either side of the midline. The midline structures like pons, midbrain, hypophysis, medulla, etc. grow at a slower pace than the cerebral lobes. The frontal and temporal lobes grow and expand, displace each other in opposite directions but the net growth is anterior and inferior. The growth of temporal lobe is adapted by the resorption of anterior wall of middle cranial fossa, the floor of the fossa and the lateral wall. Compensatorily, the orbital surface of sphenoid and the ectocranial surface of middle cranial fossa are depository. Growth of both the temporal lobes pushes midline bone (sphenoid); the midline bony ridge is depository (Fig. 6.4). Petrous temporal are also depository. Remodeling deepens the fossa that helps to accommodate the enlarging brain. Growth of temporal lobe, attached to the respective cranial fossae by fibrous tissue, continues beyond the

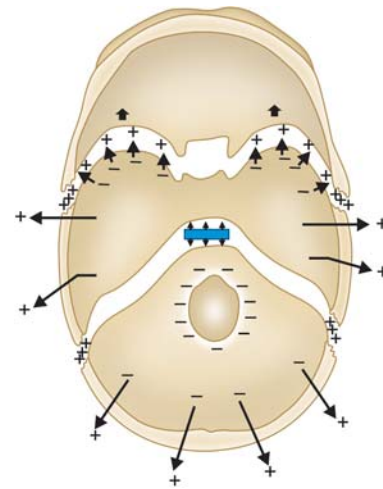


Fig. 6.4: Resorption patterns of cranial fossa with resorption and deepening of foramen magnum. Deposition takes place in the ectocranial surfaces. Proliferative activity in the synchondrosis leads to forward growth of cranial floor

period of growth of frontal lobe hence growth of temporal lobe not only pushes the middle cranial fossa outward but also anterior cranial fossa, nasomaxillary complex and mandible (Fig. 6.5). The effect appears accentuated in early life when both frontal and temporal lobes are expanding. The nasomaxillary complex is positioned in such a way that its posterior end is on the PM line, which coincides with the junction of anterior and middle cranial fossa. The complex receives positive secondary displacement due to growth of middle cranial fossa. Displacement of the cranial bones is associated with tension in the sutures and fill-in ossification. The process of secondary displacement and adaptive change in the suture ensures that the bone as a whole increases in size and relocates to a new position still maintaining its relation with adjacent bone. The sphenoidal sinus lining is resorptive, thereby increasing its size as age advances. Petrous part of temporal bone is more depository in the medial surface than the lateral. So growth of the temporal lobe causes growth of middle cranial fossa in the anterior, inferior and lateral direction. The preceding section on anterior and middle cranial fossae makes it clear that it is not just the sphenoid-occipital synchondrosis that is solely responsible for the anterior and inferior growth of cranial base. Displacement due to growth of functional matrix is an important contribution.

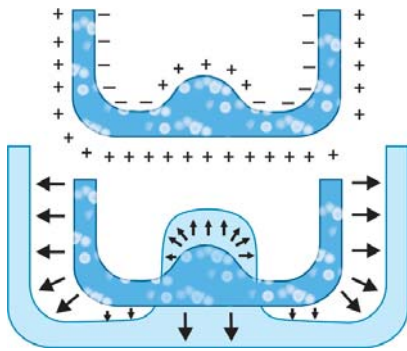


Fig. 6.5: Growth of soft tissue matrix leads to expansion of cranial floor. Expansion of cerebral lobes causes pushing up of the midline skeletal structures, and subsequent deposition in these areas. The foramina of cranial base maintain contact with their respective structures

The cranial base is perforated for the passage of blood vessels and nerves. One might wonder how the position of blood vessels with respective lobes is maintained in the ever expanding cranial fossa. As the cranial fossa remodels, it is displaced to a new location. Thus, the position of nerves and blood vessels are maintained (Fig. 6.5). The posterior cranial fossa houses the occipital lobes and cerebellum. One of the most important structures in the midline of the posterior cranial fossa is the clivus. It is a bony incline with synchondrosis. It is the inclination of the clivus that is responsible for anterior and forward growth of the cranial base. It displaces the nasomaxillary complex downward and forward. Expansion of cerebral lobes causes displacement of occipital bone similar to that in the anterior and middle cranial fossa. There is tension adapted bone growth at the sutures. The extensive cortical remodeling deepens the posterior cranial fossa and relocates the foramen magnum downwards.

A *synchondrosis* is a cartilaginous immovable type of joint where hyaline cartilage divides and is subsequently converted into bone. In the cranial base, four types of synchondroses are seen. They are intersphenoidal, interethmoidal, sphenoethmoidal and sphenoccipital or basioccipital. Synchondroses are remnants of cartilage from the prenatal life. Synchondrosis is like two epiphyseal plates juxtaposed against each other so that growth at the synchondrosis will be twice as much as it is in the epiphyseal plates.

Sphenoccipital synchondrosis starts to fuse by 13-15 years of age and by 20 years it is completely fused. The interoccipital synchondrosis closes at the fifth year, and the intersphenoid at birth. Sphenoethmoidal synchondrosis fuses at 5-20 years of age. Growth of cranial base was previously attributed solely to synchondrosis. Postnatally, anterior cranial fossa completes its growth by 8-9 years of life. In other words, sphenoethmoidal synchondrosis also fuses around the period of mixed dentition. It is only the sphenoccipital synchondrosis that is patent for enough growth to be expressed.

Synchondrosis is composed of a central layer of small cartilaginous cells, with proliferative zone, hypertrophic zone and zone of endochondral ossification on either side. Advance in age has its effect on the synchondrosis as well. The central layer of cells is wide and highly cellular, very regular in arrangement and perpendicular to the long axis of clivus with elongated cells at birth. With age, the cellularity is reduced. There is an increase in width of proliferative and hypertrophic zone till 3 to 4 years of age, after which it reduces. The number of cells in the proliferative zone becomes scant at the period of fusion of the synchondrosis. The change in hypertrophic zone at this stage is the loss of regularity of cellular arrangement. Bundles of collagen fibers are present in the perichondrium which increase in density by 6 to 7 years of age. The upper part of synchondrosis is very fibrous with a minimum number of cells. Endochondral ossification starts from all the corners of the synchondrosis, anterior, posterior and lateral. The ossification starts by 12 to 13 years of age, but the entire synchondrosis is not ossified before 16 to 17 years of age. There is a stage in the ossification of hyaline cartilage described as *asbestos transformation* which heralds the degeneration of synchondrosis. Asbestos formation is a sign that growth has ceased and ossification is about to start. Bridging of the synchondrosis occurs within a year of asbestos formation. All the cellular changes are evident in the anteroposterior direction of the synchondrosis while the endocranial surface of the clivus itself is resorptive due to pressure applied by the growing brain. Deposition occurs in the ectocranial surface of the cranial base. A study by Thilander and Ingervall (1973) showed that there is growth cartilage in the sella turcica which is patent till the third year of life. Synchondrosis is a primary cartilage and has been described as a growth

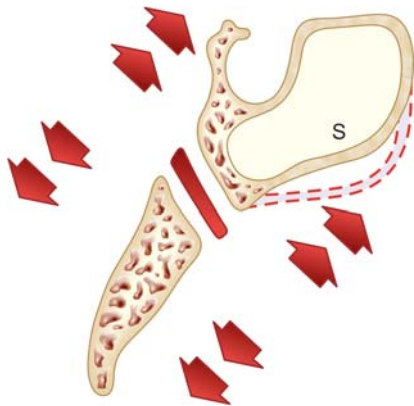


Fig. 6.6: Growth at synchondrosis with tissue separating force

center till now. Growth center is one that generates tissue separating force by its growth (Fig. 6.6). Transplantation experiments with synchondrosis have proven them to have genetic potential for independent growth but it is not as potent as an epiphyseal plate. Synchondrosis bears the weight of growing brain, growth at synchondrosis is pressure adapted like any other cartilage. On the contrary, the expanding lobes cause displacement of bone which initiates tension in the sutures. Thus, sutures have tension adapted bone growth and cartilages have pressure adapted bone growth.

Clivus undergoes resorption at the endocranial side and deposition of the ectocranial side that lengthens it and leads to an anterior and inferior drift. Growth at synchondrosis also leads to an anterior and inferior displacement. This secondarily displaces the nasomaxillary complex downward and forward. The downward displacement of nasomaxillary complex causes vertical growth of the middle and lower face.

Growth in the length of the clivus may be due to lengthening at the sphenoccipital synchondrosis. The posterior cranial fossa including the rim of the foramen magnum undergoes resorptive remodeling that lowers all these structures. Length of the cranial base at birth is about 63 percent of adult size, by first year it is about 83 percent complete and by 15 years, 98 percent complete.

Summarizing the growth of cranial base, increase in size of the cranial base is due to primary displacement of bones due to growth of functional matrix, i.e. lobes of brain and linear displacement caused by growth at synchondroses mainly sphenoccipital. The displacements

are associated with a tension related bone apposition at the sutures. There is also extensive cortical remodeling of cranial base with resorption and deepening of the fossa and deposition of bone at the ectocranial surface.

NASOMAXILLARY COMPLEX

Comprehension of postnatal growth of maxilla and mandible is made easy after the basic concepts of growth are understood. There are two basic growth movements, drift and displacement. Drift is otherwise called cortical remodeling. It is achieved by selective apposition and resorption of cortical surfaces (both endosteal and periosteal). Displacement, movement of the entire bone, in turn is classified as primary and secondary displacement. Primary displacement (translation) is the movement of bone due to its own growth. Transformation is cortical remodeling told otherwise. According to Moyers, the nasomaxillary complex functions are:

- To provide the airway;
- By housing the alveolar process of teeth to occlude and masticate;
- To enclose the sinuses, thus reducing the weight of the skull and adding resonance to the voice;
- To lodge the olfactory nerve and thereby the sense of smell;
- To condition the inspired air by its mucosal lining.

Many bones of craniofacial skeleton grow according to Enlow's expanding 'V' principle. Imagine the bone to be an ever expanding V. Bone apposition takes place on the inner side of V and resorption takes place on the outer surface. As the V expands, the inner and outer portions not only come to occupy new positions but also the bone as a whole has increased in size.

During bone growth by primary displacement, the entire bone is relocated to a new position but resorbed at the surface in the direction of growth (anterior surface for maxilla), there is bone apposition at the posterior end to maintain contact with adjacent bone. This is explained using a schematic diagram by Enlow and Bang in which a man is pulling a cart with a brick wall on it. As the cart is moved forward, the surface of the brick wall is demolished in the direction of movement and constructed in the opposite direction. Thus to quote Enlow, "as bone grows by surface deposition in one direction, it is simultaneously displaced in the opposite direction".

Area relocation theory by Enlow and Bang states that "specific local areas come to occupy new actual positions in succession, as the entire bone enlarges. These growth shifts and changes involve corresponding and sequential remodeling adjustments in order to maintain the same shape, relative positions and constant proportions of each individual area in the maxilla as a whole".

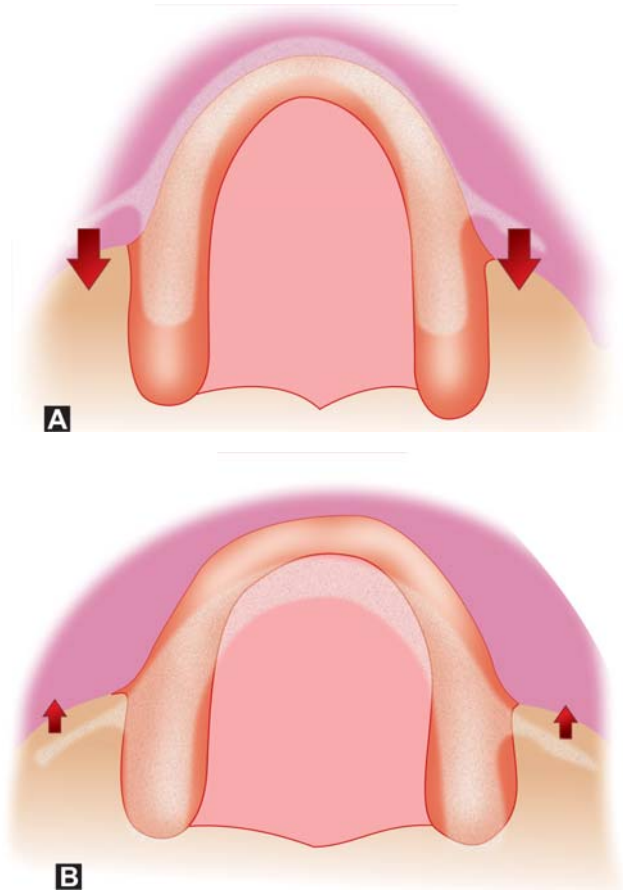
Maxilla cannot be considered as a separate bone; instead its growth is best studied, taken into account the whole nasomaxillary complex or midface. It is a complex system of sutures through which all the bones are in contact. The sutures are zygomatico-maxillary, zygomatico-temporal, zygomatico-frontal, fronto-maxillary, nasomaxillary, frontonasal, intermaxillary, etc. The nasomaxillary complex consists of zygomatic bone, maxilla (with palate), nasal bone; part of frontal (orbital roof) bone.

Motive force behind the growth of maxilla has been attributed to primary displacement, growth at synchondroses, sutures, septal cartilage, etc. Primary displacement of maxilla is due to growth of maxillary tuberosity (Figs 6.7A and B). The tuberosity is considered as a major growth site. Cortical deposition at this site pushes against the posterior structures with a counter anterior thrust that leads to primary displacement. The accepted fact here is that bone is pressure sensitive and succumbs to pressure. The posterior growth only helps to lengthen the dental arch of maxilla.

Synchondrosis at the cranial base especially sphenoccipital synchondrosis grows to lengthen the cranial base. This provides an anterior thrust to the midface (Fig. 6.8). As the cranial base grows anteriorly and superiorly, the midface grows anteriorly and inferiorly. This is termed secondary displacement. The midfacial bones, by cortical apposition at the posterior end, reestablish contact with the cranial base. The upper face grows upward and forward and lower face grows downward and forward as an expanding V.

Sutural theory proposes that the sutures of the nasomaxillary complex are centers of growth. Proliferation of osteogenic tissue at the sutures causes growth movement that pushes the bone apart with later fill-in. We know that sutures are pressure sensitive; hence sutures can act only as fill-in areas in secondary displacement but cannot provide the force for primary displacement of bone.

Nasal septal cartilage growth can lead to the anterior growth shift of the complex. The theory by Scott that



Figs 6.7A and B: Depiction of posterior growth at tuberosity (A) and anterior displacement (B)

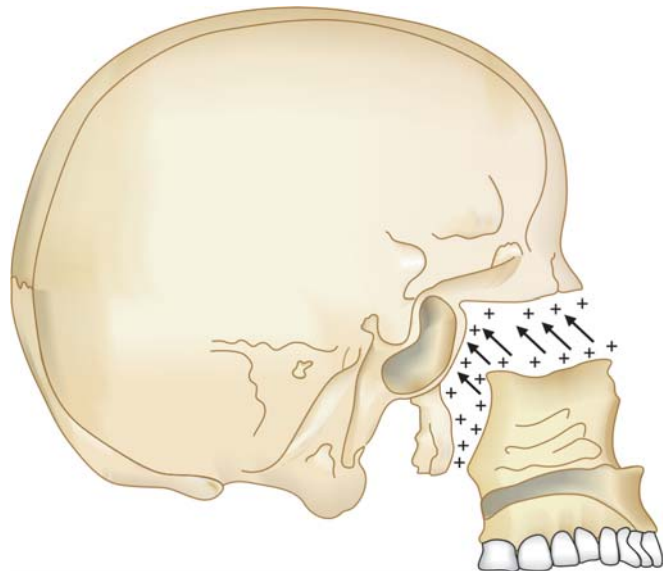


Fig. 6.8: Thrust of nasomaxillary complex downward and forward with secondary adaptive deposition at the sutures

claims nasal septal cartilage to be growth center has been accepted. Growth of cartilage leads to anterior and inferior displacement of the complex, splits the suture, tension in the sutural area leads to bone fill-in. Growth of functional matrix (soft tissue) also can secondarily displace maxilla in space, with adaptive deposition at sutural margins.

Nasomaxillary growth can be studied regionally at best. The dimension of face that completes growth early is width, followed by depth and adult height is achieved last. At birth, the height difference between cranium and face is 8:1. As the child grows, the ratio drastically changes due to increase in depth and height. At birth, the length of face is equal to width. Increase in length is due to inferior displacement of bone and alveolar growth. Width of the face follows the neural curve in Scammon's curve whereas depth and height follows the general body growth.

Maxilla

The two maxillae articulate with each other in the midline at the intermaxillary suture. As already mentioned, maxillae are attached to other bones by a complex sutural system. Maxilla grows downwards and forwards in response to various forces. It is a surprising fact that as maxilla grows forward, the posterior end is depository to maintain contact with adjacent bones but the entire anterior surface of maxilla becomes resorptive to maintain the shape and configuration. Postnatal growth of nasomaxillary complex was extensively studied by Enlow and Bang and their study forms the basis to understand the topic. Bone deposition is seen at the entire inner aspect of the maxillary arch and at the tuberosity. At the anterior concave surface of maxilla, the periosteal concavity from ANS to point 'A' is depository and the periosteal surface from point 'A' to alveolar margin is resorptive (Fig. 6.9). The reverse occurs in the endosteal side of cortex, upper half resorptive and lower half depository. The key ridge is an important site of reversal and remodeling. The anterior surface of maxilla till the region of key ridge is resorptive and is concave, facing downwards and growing inferiorly. It is at the region of key ridge (approximately first molar region) that reversal occurs (Figs 6.10 and 6.11), the lateral surface of maxilla posterior to key ridge and lateral surface of tuberosity are depository, growing laterally, facing upward. The medial surface of zygomatic arch is resorptive and lateral surface depository as compensation. Analogy of

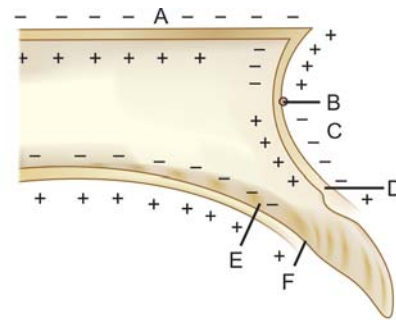


Fig. 6.9: Remodeling pattern of anterior surface of maxilla

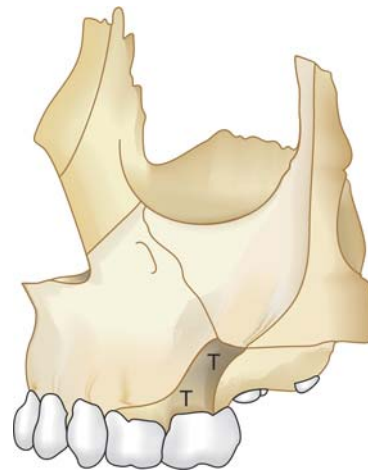


Fig. 6.10: Darker shaded areas are depository and lighter shaded areas are resorptive

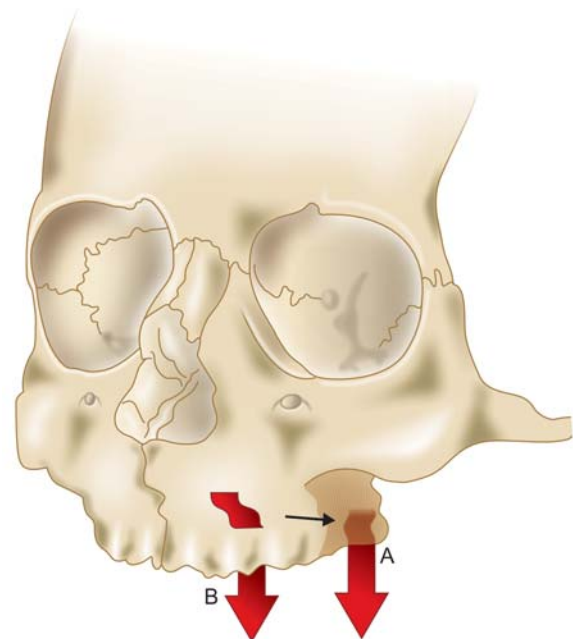


Fig. 6.11: Difference in remodeling in the anterior surface of maxilla, reversal is at key ridge. Part in area 'B' is concave and resorptive while part 'A' grows by periosteal deposition

expanding V implies that maxilla grows inferiorly due to deposition on the inner aspect of maxillary arch and palate and resorption in the outer aspect.

The frontal process of maxilla and nasal bone that form the bridge of the nose are depository in the anterior aspect. This facilitates forward placement of the medial part of the face compared to the lateral aspect. The medial rim of orbit is depository and the lateral rim resorptive that accentuates the condition. Pyriform rim is resorptive. Thus, the remodeling pattern of maxilla is so complex that there is an extensive variation in the anterior surface alone. Though the bridge of the nose receives deposits in the anterior surface, the width of bridge does not vary much with age. This aspect of the bridge is determined early in life and there is no great change in the distance between the inner canthus with growth.

As the maxillary dental arch is lengthened by deposition posteriorly at the tuberosity, the lateral surface also undergoes deposition. The maxillary sinus is depository on the medial surface and resorptive in all the other surfaces. This is selective remodeling as a compensation for the laterally expanding nasal fossa. The lengthening of dental arch allows space for the eruption of all the molars. The location of tuberosity is marked by the posterior limit of anterior cranial base. This is called Posterior Maxillary (PM) plane. The position of posterior limit of anterior cranial base, maxillary tuberosity and junction of corpus and ramus of mandible (lingual tuberosity) are all on the PM plane at the end of growth according to Enlow's counterpart principle/principle of growth equivalents.

The vertical growth of maxilla is due to inferior displacement and adaptive apposition at the sutures. The alveolar margin of maxilla undergoes enormous amount of growth with eruption of teeth. The downward displacement (primary and secondary) of maxilla and mandible increases the interocclusal space, enough for the alveolar growth and eruption of teeth. Height is the last dimension to complete growth. Increase in height of alveolar margin accompanies eruption of teeth. Eruption of teeth is different from vertical drift. Vertical drift is the movement of tooth, socket together. During eruption of teeth, there is formation of root that drives the teeth to erupt. In drift, periodontal tissue also undergoes extensive remodeling. Vertical drift can be used to treat cases by working with growth, relative

intrusion is an example. Primary vertical displacement of maxillary complex is accompanied by extensive remodeling on the periosteal surface (deposition) and resorption at the endosteal surface. There is concomitant bone formation at all the sutures. The growth at sutures can cause slippage of suture against other sutures which is corrected by remodeling changes. Enlow states that lacrimal suture acts as a key growth mediator as it provides for the slippage of the multiple bones. Lacrimal bone functions as a hub for key traffic control in development therefore without the 'perilacrimal sutural system', a developmental gridlock will result.

Thus downward increase in height of alveolar housing may be due to:

- Tooth eruption.
- Vertical drift of teeth.
- Passive movement of dentition along with maxilla.

Palate

Downward drift of palate is extensive. The shallow palate of the new born is not retained in the adult. There is enormous change in both size and shape of the palate with growth. The newborn's palate is shallow and the horse shoe shaped dental arch has equal length and width. As age advances, the palate receives extensive deposition at the roof. This is part of the remodeling of the face. The nasal floor is resorptive, nasal roof is depository. The length of nasal floor is increased. Concomitant with the resorption of nasal floor, palatal roof receives bone deposition (Fig. 6.12). Palatal growth can be explained with the help of expanding V, deposition on the inner aspect of V (palatal roof) and resorption on the outer aspect (nasal floor) expands the V in the direction of open end. The eruption of teeth increases the vertical height of alveolar bone and depth of palate, it increases the width of the bone laterally according to V principle; palate grows in height and width with the leading surface towards growth undergoing deposition. This increase in width by maxilla due to V principle is evident. Increase in width also receives contribution by apposition at the intermaxillary suture. Midpalatal suture, according to Enlow, contributes only a meager amount to increase in width, so theoretically speaking slow dentoalveolar expansion should provide the same level of stability as RME and slow expansion can actually increase arch width. There is no need for sutural split.

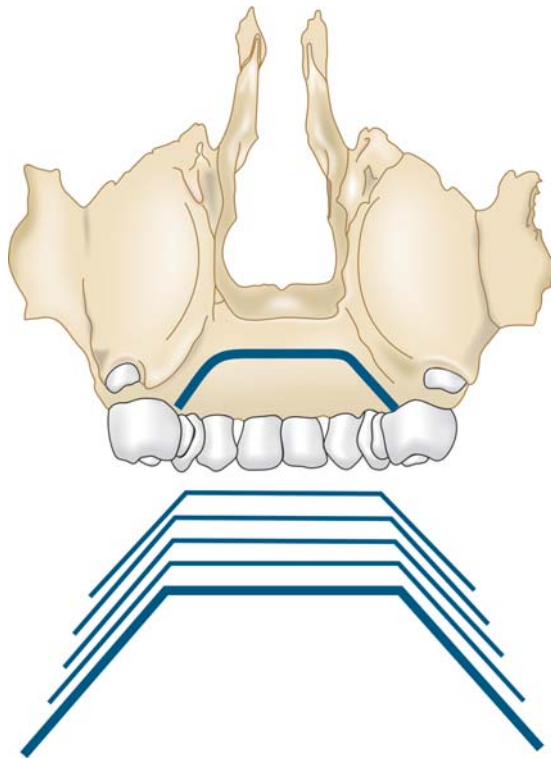


Fig. 6.12: Downward growth and expansion of palate in the form of V due to deposition at the palatal roof

Zygomatic Bone

As the maxilla is displaced anteriorly, its anterior surface is resorptive, the zygomatic bone shifts posteriorly. The anterior surface of the zygomatic bone and medial surface (temporal) are resorptive just like maxilla. The posterior and lateral surfaces are depository (Fig. 6.13). This expands the zygomatic bone bilaterally, and bizygomatic width increases with age. The bone as a whole, relocates posteriorly and remodeling just augments it in such a way that the bony prominence of cheek comes to be directed laterally as the child grows. The cheek bone becomes broad. The temporal fossa increases in volume. Though all the remodeling changes of anterior surface of face are indicative of a resorption pattern, the primary displacement of individual bones is downward and forward (anterior). The remodeling changes are just to maintain the shape and proportion. To counter anterior displacement, there is apposition at the zygomaticofrontal and zygomaticotemporal sutures.

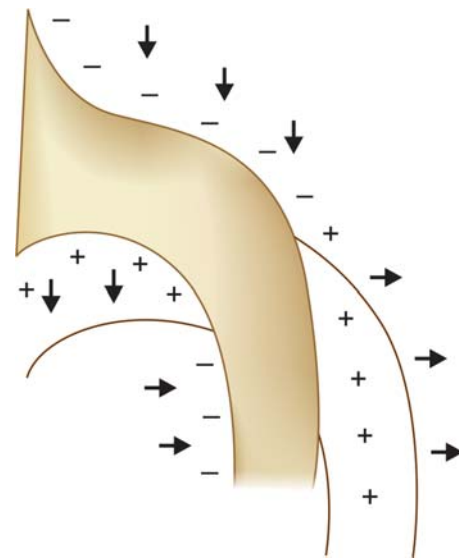


Fig. 6.13: Remodeling of zygomatic bone. Resorption is evident in the anterior and medial surface and deposition at posterior and lateral surface. Zygoma points laterally, there is increase in interzygomatic width

Nasal Cavity

The floor and lateral walls of nasal cavity are resorptive with deposition in the medial wall of maxillary sinus. This expands the nasal cavity. The portion of roof near the olfactory fossa is depository because endocranial surface is resorptive. This remodeling pattern lowers the roof of the nose. In turn, the floor of the nose is lowered by resorption and concomitant deposition on the palatal side (Fig. 6.14). The maxillary sinus is resorptive in the lateral wall and depository in the medial wall. The conchae are remodeled by deposition in the lateral and inferior side and deposition on the superior and medial sides. The width of bridge of nose does not vary to a great extent. With remodeling, the pyriform aperture is widened.

Orbit

The orbit is a complex congregation of bones. The orbit has medial and lateral walls, roof and floor. In the medial wall of the orbit, lacrimal and ethmoidal bones are present. As the nasal cavity elongates, medial wall of orbit receives deposition; it also expands laterally. The lacrimal bone and the sutural system surrounding it,



Fig. 6.14: Remodeling of orbit and nasal cavity

according to Enlow, are the key for midfacial growth and development. The phenomenon of sutural slippage has been explained. Many bones of nasomaxillary complex expand, grow and displace at different junctures. It is the lacrimal sutural connective tissue which allows for slippage of the bones at the interfaces. The slippage is probably responsible for net downward and forward growth of nasomaxillary complex. The slippage is due to the presence of myofibroblasts which are contractile cells that provide the tension in the sutural system with bone displacement, this pulls one bone across the suture towards another. "One bone slides along the suture as new bone tissue is laid down at suture margins". The ethmoidal air sinus is also enlarging. The roof of orbit is the floor of anterior cranial fossa and this endocranial surface is resorptive to accommodate the growing frontal lobe. Compensatory deposition occurs in the orbital roof to keep this already thin bone intact. This remodeling relocates the roof anteriorly and inferiorly (Fig. 6.14). Similarly, the floor of orbit also receives deposits of bone. The floor faces laterally and deposits on this surface make it face laterally. The lateral wall of orbit undergoes resorption in the medial surface and deposition in the lateral surface, thereby drifting it outward. Orbit expands by V principle. There is deposition on the inner aspect and selective resorption on the outer aspect. The supraorbital ridges are depository but the area below and lateral to it, the anterolateral rim of supraorbital rim is resorptive.

Orbit and the nasal cavity share a very interesting relationship. Eyeball follows the neural growth of Scammon's curve; infact, eyeball grows massively in the prenatal life more than the frontal lobe. The lower border of orbit is almost in line with the nasal floor vertically at birth. With growth, the remodeling of nasal floor increases the height of nasal cavity, but since eye completes its growth first, there is early cessation of growth, thus the difference in height between the nasal floor and infraorbital rim. All the bones of the face are secondarily displaced in a downward and forward direction. Though displaced downward, orbit is simultaneously moving away by deposits in the floor. Thus, different parts of the same bone, orbital surface of maxilla and nasal floor are moving in opposite directions with growth.

Another surprising aspect of midfacial growth in the behavior of bones is that the bony parts in the center of the face like nasal bridge, medial rim of orbit are getting deposits and growing forward with growth but those in the lateral aspect of the face (lateral orbital rim, pyriform rim) are not only remodeled laterally by resorption but also posteriorly. Thus, the face that was flat at birth has developed a configuration wherein the central aspect of face is more anterior to the lateral aspect and there is a gradual slope from medial to lateral. This is pictured as accentuated by the unique remodeling of zygomatic bone that is moving both laterally and posteriorly by resorption in the anterior periosteal surface and deposition on the lateral periosteal surface. The lateral aspect of supraorbital rim is also resorptive. All these changes help to increase the width of the face.

There is a similar hierarchial arrangement of bones by remodeling in the superoinferior direction. The face appears to point anterior superoinferiorly at birth. There is deposition in the superciliary arches and resorption of the infraorbital rim that drifts it posteriorly. There is concomitant resorption of pyriform rim to drift it posteriorly. All these changes lead to a backward cant of the face in the superoinferior direction in an adult. To add to all this, the anterior surface of maxilla is resorptive and it is also displaced and remodeled downward.

MANDIBLE

Mandible is a unique bone, both by its structure and function. It is a horse shoe shaped bone with vertical

ramus at the end of the horseshoe. It houses the only movable joint of the skull at both its ends. Mandible has a corpus, two ramii, two coronoid and two condylar processes. It holds the lower set of teeth in its alveolar process by means of gomphosis. Mandible in other mammals is a bone with a long snout, so is maxilla. There is an enormous growth of forebrain, frontal lobe with evolution. Man owes the presence of forehead to the growth of frontal lobe. Cerebrum as a whole is longer in man, growth of temporal lobe leads to redirection of eyes from lateral to frontal aspect. The growth of forebrain leads to flexure of the olfactory lobes from the vertical to horizontal position. By the laws of nature, the nostrils come to occupy a position perpendicular to that of olfactory vesicles, hence nasomaxillary complex comes to be directed downward and, middle and lower face are tucked under the head. The mandible is also reduced in size, ramal angle is decreased and ramus height increases. Functions of mandible include (i) providing mobility to the jaws by the TMJ; (ii) mastication by teeth and are the site of insertion of muscles of mastication; (iii) maintenance of airway, ramal width coinciding with pharyngeal width.

Mandible, at birth is small, with short ramus, large gonial angle, and flat mandibular fossa with no articular eminence. The condyles are at the level of the occlusal plane. Mandible is formed of numerous micro skeletal units, alveolar, condylar, coronoid, ramus, symphysis etc. Mandible is the best example to explain expanding V principle. It is not just due to the shape of the bone. Every part of the bone undergoes remodeling following the expanding V principle, viz apposition on the inner aspect of V that is growing towards the direction of growth and resorption on the outer aspect (Fig. 6.15); not only expands the V but there is also growth at the ends of the V; there is increase in the length of the bone as well.

Growth of the mandible was thought to occur principally by growth at condyle. Superior and posterior growth of condyle presses against the glenoid fossa/cranial base (cartilage has pressure adapted bone growth) which provides an anterior thrust to displace the lower jaw forward. This posterior growth and anterior displacement is akin to that described in the growth of maxilla. Ranly explains the concept with the example of a man swimming. At the start of the swimming process, the legs are pressed against the wall of swimming pool.

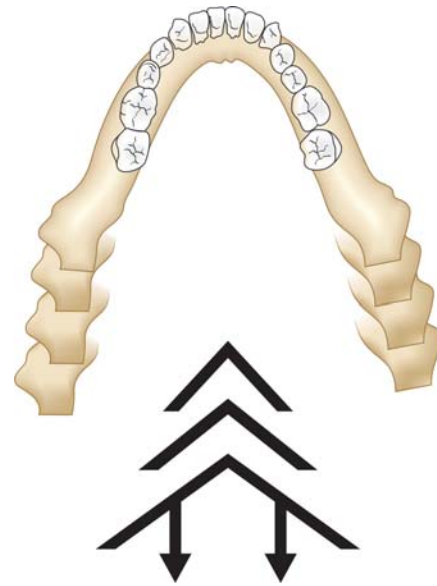


Fig. 6.15: Growth of mandible according to V principle

This pressure gives a thrust to the swimmer to surge forward. The concept of posterior growth and anterior displacement leads to primary displacement. Moss considers that it is not the growth of condyle that leads to anterior displacement, instead, the expansion of orofacial capsule leads to passive displacement of mandible with secondary adaptive growth in the condyle. Growth of mandible can also be due to growth at synchondrosis (though not accepted as it is for maxilla) which pushes the face anteriorly and inferiorly. Ultimately, anterior and inferior displacement leads to separation of maxilla and mandible to provide for enough interocclusal space. There is also a definitive increase in arch length by ramal remodeling posteriorly to maintain condylar contact with temporal fossa.

Increase in the length of mandibular corpus occurs by resorption in the anterior border of ramus. This allows the growth in length of dental arch to accommodate the permanent molars. The earliest concept of corpus lengthening stated that there is resorption at the anterior border of ramus and deposition at the posterior border so that ramus is shifted to a more posterior location and corpus lengthened (Fig. 6.16). This concept was proposed by Hunter. Later, it was found that mandibular growth cannot be simplified into an anterior resorbing and posteriorly depository ramus. Mandible undergoes a rotational pattern of growth.

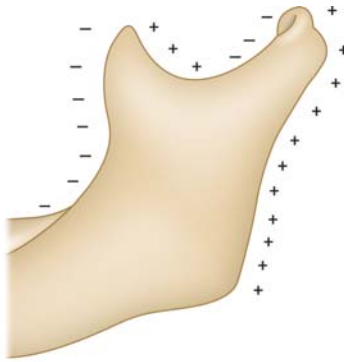


Fig. 6.16: Ramal remodeling: Hunterian concept

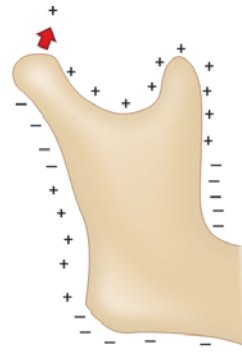


Fig. 6.17: Remodeling associated with ramal uprighing

The remodeling of ramus occurs in an arcial pattern. With anterior displacement, condyle maintains contact with the temporal fossa. The ramal angle of childhood slightly uprighs in adolescence and in late adulthood, it becomes acute. Till the uprighing of ramus, there is deposition along the posterior ramal border, but after uprighing, there is selective deposition/resorption pattern in the posterior and anterior borders. Inferior part of anterior margin is resorptive whereas superior portion is depository. On the contrary, the inferior portion of posterior border is depository and superior portion is resorptive (Fig. 6.17). The anterior margin of coronoid process also is depository so that the ramus appears to have rotated slightly to change the angulation though it is in the same position. There is not only change in angulation of ramus but there is also an increase in vertical height of ramus. The gonial angle closes and is shifted to a posterior position. On the whole, the ramus appears to have rotated around an arc (Fig. 6.18). The breadth of ramus remains the same. Increase in breadth of ramus is seen only till there is enlargement of pharynx and middle cranial fossa (Enlow's counterpart principle). With the remodeling of ramus posteriorly, the mandibular foramen maintains its position by deposition in the anterior rim and resorption in the posterior rim (Fig. 6.19); it also shifts posteriorly and is thus always centered in the medial surface of the ramus.

The coronoid process has a twisted form (propeller like twist as described by Enlow). The medial surface of the process faces posteriorly, superiorly and lingually all at one time. Deposits on the medial surface of the coronoid lead not only to posterior lengthening of the

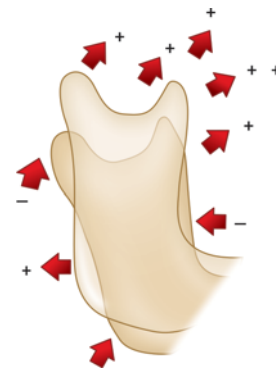


Fig. 6.18: Ramus: Uprighting and direction of rotation

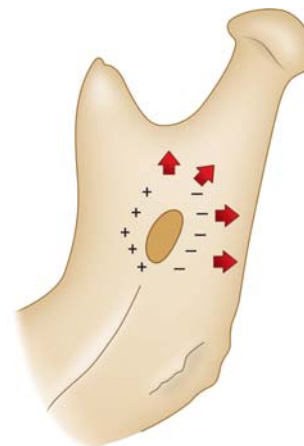


Fig. 6.19: Mandibular foramen relocates posteriorly to maintain its position in the ramus

mandible (V principle) but also an increase in height occurs (Fig. 6.20). When the sections of the region of coronoid process are taken and bone at various stages of development superimposed, the coronoid process is seen to grow in length, with increase in thickness due to deposit on the medial side; coronoid also becomes posteriorly relocated. There is resorption on the buccal surface of the coronoid process. The buccal surface of the process faces away from all the three directions. There is distinct difference in the direction of orientation between the medial surface of the coronoid process and ramus. The area below the depository surface of the coronoid process (medial surface of ramus) is resorptive while the buccal surface of ramus is depository (Fig. 6.21). The buccal surface of ramus faces posteriorly towards the direction of growth. Due to backward growth (combined with expansion like V) of mandible, the area that was occupied once by ramus and coronoid process, comes to be occupied by the lingual tuberosity. There is increase in both length and width of mandible.

The corpus or body of mandible is depository on the outer surface and resorptive on the inferior aspect of the medial surface (Figs 6.21 and 6.23). The superior aspect of the medial surface just below the teeth is depository. In the ramus, remodeling on the medial surface of ramus follows the same pattern as in the corpus. Now, viewing the medial surface of the ramus, it is seen that the remodeling is in the form of 'L', with the depository area extending from the superior half of medial surface of corpus to the anterior half of medial surface of the ramus (below coronoid). The resorptive area follows depository area, from inferior half of medial surface of corpus to posterior half of medial surface of ramus (below the condyle, Fig. 6.22). This eccentric remodeling is to achieve the configuration of adult mandible. The dental arch comes to be directed towards the midline of the mandible. Increase in height of alveolar bone accompanies eruption of teeth. With the descent of the maxilla and separation of two bones, the mandibular anterior teeth erupt superiorly and lingually. Similar to maxilla, mandibular width completes first, followed by depth and height.

The remodeling changes in the symphysis are very unique and the chin is a characteristic very special to humans. The long snout of other mammals is redirected to vertically oriented upper and lower jaws, nevertheless a memory of the snout is retained in the form of the chin.



Fig. 6.20: Coronoid process as an expanding V. Grows medially and vertically



Fig. 6.21: Apposition/resorption pattern of mandible

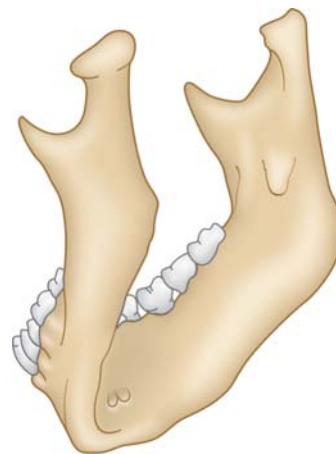


Fig. 6.22: Lighter areas are depository and darker areas are resorptive



Fig. 6.23: Blue arrows resorption, red arrows deposition

The remodeling pattern tries to accentuate the prominence of the chin. There is deposition on the chin itself while the area of anterior surface of alveolus above the chin is resorptive (Fig. 6.25). There is corresponding deposition in the endosteal surface. The lingual periosteum of the symphysis is depository (see Fig. 6.24). Resorption of bone at the anterior alveolus thins the bone on the surface of roots of lower anterior teeth. Danger of exposure of root is avoided by gradual uprighting of lower incisors that happen with age. Lower border of mandible is depository except at the antegonial notch.

Growth at the condylar cartilage is pressure adapted. Superior surface of condyle is depository. Only the cap of condyle undergoes endochondral ossification, the rest of the condyle and the neck of condyle grows by intramembranous ossification (cortical remodeling). The condyle grows like an expanding V. There is deposition on the inner aspect of V and resorption on the outer surface (Fig. 6.26). The neck of condyle is resorptive on the buccal and lingual surfaces and this, coupled with deposition on the condylar head, contributes to the V configuration. The buccal and lingual surfaces of the neck are equally resorptive throughout; the inferiorly facing end of buccal surface and superiorly facing end of lingual surfaces are depository (Fig. 6.27). The cross-section of condylar neck is like a tear drop. Imagining the remodeling of neck in this shape makes visualization of remodeling easier. The region that was once condyle is gradually remodeled to a neck, condyle relocates to a more posterior and superior position. Resorption of the condylar neck on the periosteal side is accompanied by deposition on the endosteal surface.

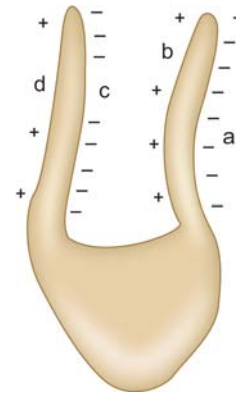


Fig. 6.24: Symphysis. Deposition on the lingual surface and resorption on the labial alveolar surface

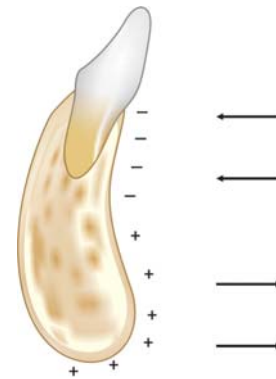


Fig. 6.25: Deposition on the chin to accentuate the prominence

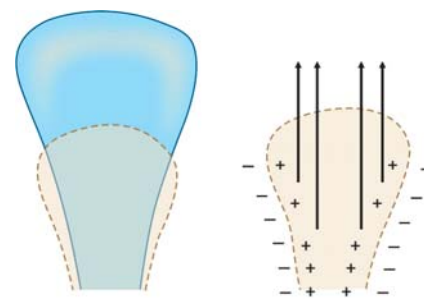


Fig. 6.26: Condyle as an expanding V with deposition on the inner aspect and resorption on the outer aspect of V

Condylar cartilage was once thought to be the soul of mandibular growth, as the responsible growth center. It is now a known fact that the condylar cartilage is not a primary cartilage but just a secondary cartilage.

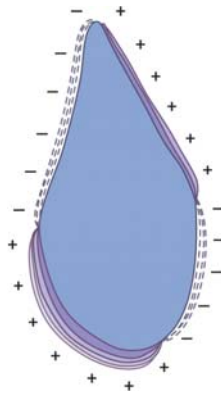


Fig. 6.27: Condylar neck. Both resorption and deposition occurs on the buccal and lingual surface

According to Petrovic, the secondary cartilage is more open to external forces. It can be manipulated by external environmental influences. In the secondary cartilages like condyle, the zone of growth contains proliferative cells like skeletoblasts and prechondroblasts. They do not secrete cartilaginous matrix, the cells of the this zone are just surrounded by type I collagen unlike in primary cartilage where the cells are surrounded by cartilaginous matrix. The cells of zone of growth in secondary cartilage, thus, are exposed to the environment and are moldable to external influences. This is used to advantage in functional treatment. Condylar cellular arrangement also is to the orthodontist's advantage. The cells of condylar cartilage are not arranged in rows as it is in primary cartilages. The condylar cartilage has multidirectional proliferative capacity. The condyle can remodel superiorly and posteriorly at the same time.

Lingual tuberosity is a very important anatomic site in mandible at the junction of corpus and ramus at the medial aspect. Lingual tuberosity is the counterpart of maxillary tuberosity. Deposits on the tuberosity will cause a definitive posterior growth of the posteriorly facing tuberosity (Fig. 6.28). It not only faces posterior but also is oriented towards the midline than the ramus. If viewed from the occlusal aspect, lingual tuberosity appears to be in line with the dental arch whereas ramus is slightly away along the arms of the expanding V. The region below lingual tuberosity is resorptive thereby accentuating the prominence of tuberosity. When juvenile and adult mandibles are compared with the view from occlusal surface, the tuberosity is greatly relocated in a posterior

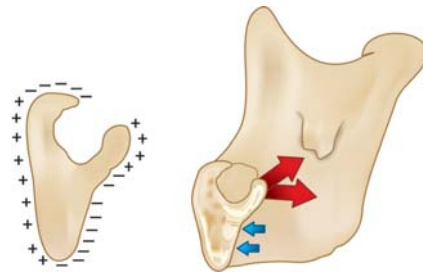


Fig. 6.28: Lingual tuberosity, depository surface

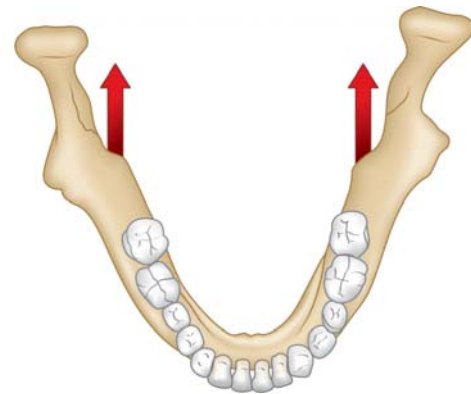
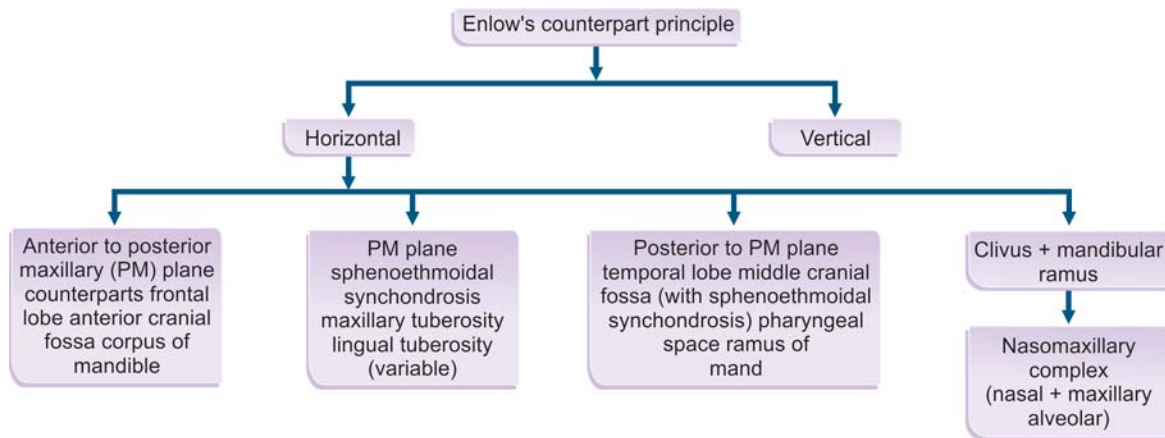


Fig. 6.29: Posterior growth of lingual tuberosity due to deposition

direction (Fig. 6.29), yet the mediolateral growth is meager when compared to the posterior shift. Enlow points out that it is due to the stable bicondylar width established early in childhood. Bicondylar width in turn is related to the width of the cranial base that completes early. The area of ramus just behind the tuberosity appears to remodel medially, yet the ramus on gross examination appears to be lateral to the lingual tuberosity.

Lingual tuberosity, as already mentioned, is a direct counterpart of maxillary tuberosity and is equally an important growth site for the mandible. When viewed from the lateral aspect, the lingual and maxillary tuberosity appear to be positioned along the same vertical line called the posterior maxillary plane or PM plane. This key anatomic plane forms the reference basis for Enlow's counterpart principle or principle of growth equivalents (Flow chart 6.1). This plane extends from the junction of anterior and middle cranial fossa and extends downward in a direction perpendicular to the vertical axis of the orbit. The structures anterior and

Flow chart 6.1: Enlow's counterpart principle



posterior to the plane of counterparts to each other. To be more explanatory, the frontal lobe, anterior cranial fossa, upper part of ethmomaxillary complex, palate, maxillary arch and corpus of mandible are counterparts anterior to PM plane. The posterior boundary of all these structures is placed on the PM plane. Thus, maxillary and lingual tuberosities that form the posterior boundary of maxilla and corpus of mandible are usually on the PM plane. Mandible is an independent bone and it does not undergo the same growth process as all the other bones of the face. The midface and upper faces are more closely related to the bones of the cranium than the mandible is. So, the posterior boundary of mandibular corpus falling exactly on PM plane is subject to variation. According to Enlow, it is not just the lingual tuberosity that falls on the PM plane but also the anterior edge of ramus of mandible. In deviations, the anterior border may be in front or well behind the PM plane. Mandibular growth is subject to variation like rotational growth of mandible, physiology of muscles of mastication, pathologies of the TM joint, etc. So, the lingual tuberosity may not always be on the PM plane. It may be subjected to great variation. In average growing persons with acceptable facial dimensions, the lingual tuberosity is usually on the PM plane. The structure posterior to the PM plane that are counterparts of each other are the temporal lobe, middle cranial fossa, oropharyngeal space and ramal width. The enlargement/growth of oropharyngeal space is dependent on the function (respiration). With increase in anteroposterior dimension of oropharyngeal space, ramal width increases by

remodeling (as mentioned above) to keep pace with its counterparts and also to maintain the boundaries.

It is surprising to realize that the PM plane is not just an anatomic boundary but a functional one too. What makes it extremely valid as a reference line is the fact that all the other reference lines (e.g. SN plane) in cephalometrics fail to encompass the important sites. The importance of PM plane are: (i) it is a boundary between important growth controlling functional matrices (temporal and frontal lobes—neurocranial capsule, oropharyngeal spaces, etc.) (ii) it is a boundary for growth sites of the upper and lower jaw, (iii) it is a structural, developmental, morphological and functional boundary, (iv) it makes relating various structures both anatomic and functional an easy task.

Growth of anterior cranial fossa is equalized by corresponding elongation of the nasomaxillary complex. The posterior growth of maxilla is denoted by posterior arrows but the displacement is anterior. There is a concomitant increase in length of corpus by ramal remodeling. Growth of middle cranial fossa (lengthening at sphenooccipital synchondrosis) is associated with enlargement of pharyngeal space and subsequent increase of ramal width. The sphenoethmoidal synchondrosis appears to be a counterpart of maxillary tuberosity and is on the PM plane. These structures are vertical counterparts of each other as well, vertical lengthening of clivus and mandibular ramus are counterparts to vertical lengthening of nasomaxillary complex (nasal + maxillary alveolar region). Mandible grows superiorly (at the condyle) but is displaced inferiorly.

The angle of mandible, as already mentioned becomes upright with age and subsequently becomes more acute. When the rotational pattern of ramal uprighing takes place, the region of the gonial angle is appositional and superior portion of posterior border is resorptive. The region of gonial angle receives insertion of masseter fibers. There has been widespread speculation that the areas of predominant muscle pull are generally resorptive because work of the muscle is distributed to its insertional surface (periosteum) by means of tendons, and that the work (pull) of muscle will cause resorption of bone. On the contrary, apposition of bone takes place in the region of gonial angle and resorption is seen at the area anterior to it (antegonial notch). A very valid explanation for this phenomenon was given by Frost in his "law of electrogenesis". According to him, when the muscle pull is active in certain regions then surface of that part of bone becomes concave and areas without muscle attachment become convex. This alters the surface characteristics, specific signals are generated, concave surface becomes negative and convex surface becomes positive. Surface apposition is seen in concave surface and resorption at convex surface. The same concept when applied to gonial angle makes the pattern of bone growth at this region comprehensive. Pull of the masseter muscle makes the bone surface concave that leads to constant deposition at the gonial angle.

TEMPOROMANDIBULAR JOINT

Temporomandibular joint is the reason for the mandible being the only movable bone in the skull. It is a ginglymodiarthroidal joint. TMJ starts developing at the 10th week of intrauterine life. It is formed of the articular surfaces of condyle and temporal bones (mandibular fossa). The joint cavity is divided into two: upper and lower by the intervening biconcave fibrous, articular disk. The articular disk is avascular in the center and attached to the posterior eminence of mandibular fossa by means of retrodiscal pad. The upper head of lateral pterygoid muscle is attached to the articular disk (anterior end). The articular surface is lined by synovial membrane and joint cavities are lubricated by synovial fluid.

The condylar cartilage is a secondary cartilage. The primary (embryonic) joint is the articulation between malleus and incus. As the TMJ is a secondary joint

(embryologically), cartilage is also secondary. It is different from the primary epiphyseal cartilages of long bones in that the epiphyseal cartilages are growth centers rather than sites (centers are capable of independent growth). Condylar cartilage is merely a growth site, in other words, it is extremely adaptive in its growth. The condylar surface has four zones: (i) Articular zone; (ii) Proliferative zone; (iii) Hypertrophic zone; (iv) Zone of endochondral ossification. The cells of epiphyseal cartilage are arranged in parallel columns ascertaining the direction of growth of epiphyseal cartilages in long bones. In contrast, condylar cartilage cells are not arranged in such order, we cannot judge the direction of growth of condyle head. It has a multidirectional growth tendency. Another aspect of condylar cartilage is that the cartilage cells are patent post adolescence, adaptive remodeling can contribute to growth of mandible even after adolescence. In epiphyseal cartilages, growth ceases with ossification of the cartilage and fusion with metaphysis.

In infants, the condylar cartilage (all the zones) is highly vascular and cellular. Decrease in vascularity occurs with increasing age, by 5 to 6 years of age, the articular layer becomes less cellular, more fibrous (coarse and dense). The proliferative zone is formed of two layers, an outer layer formed of small cells, numerous rapidly mitotic and with little intercellular substance. The inner layer is less wide than the outer layer, formed of large round cells. These cells are not highly mitotic and have more intercellular substance, the superior-most part of condyle is the most cellular. Similarly, the zone of hypertrophy also consists of two layers, outer layer being formed of small, round cells. The cell size increases towards the inner layer and these cells are chondroblastic and innermost of these cells undergo pericellular mineralization. The mineralized zone has a constant width, and the erosion of the zone leads to replacement by bone. Active proliferation of cells is evident till 13 to 15 years of life, after which there is decrease in number of cells, mitotic index. Cartilage islands are seen in superior and anterior part of the condyle and posterior part of articular eminence at 20 years of age. The articular eminence and glenoid fossa at birth consists of zones similar to the head of the condyle. The articular layer is vascular at birth and becomes progressively fibrous. The fibers are longitudinally arranged. In the pubertal period, there is a transient increase in thickness of the

proliferative zone. There is rapid growth of condylar cartilage during the initial years of life but proliferation is not demonstrable in condyle during puberty. Instead, proliferation of cells in articular eminence and fossa is evident in the pubertal period. This disproves the well conceived notion that there is rapid growth of condyle during puberty.

The disk is initially flat and highly vascular. With ageing there is a marked decline in blood supply, the disk becomes fibrous, the central part thins with anterior and posterior parts becoming thicker. Collagen fibers become coarse and dense and get arranged in a three-dimensional network.

After 20 years, superior and anterior part of condyle and posteroinferior part of eminence retain the condylar cartilage. The best reasoning for this finding is adaptation to functional stress. Thus, condylar cartilage is not an active growth center; instead, it is a growth site with rapid growth during the initial years. In the later part of life, TMJ takes the job of resisting the stress and pressure. The development of articular eminence is also functional. There is no eminence at birth. This configuration helps in the forward movement of mandible during suckling. As the dentition develops and occlusion (particularly overbite) is established, the eminence develops to help the condyle establish its path of movement.

DYNAMICS OF FACIAL GROWTH

The study of growth and development is not complete if the dynamics of growth in orthodontics are not understood. The very aspect of an extensive study of growth and development by orthodontists is due to the fact that they are the only dental specialists who work with growth. Children, in mixed dentition stage with skeletal malocclusion can be treated by orthodontists, so are adolescents who are at the peak of their pubertal growth spurt. The utilization of growth to bring out positive result during orthodontic/orthopedic treatment forms the essence of studying growth by clinicians. The aspects of growth that needs to be discussed for clinical significance can be divided into certain concepts of general body growth and concepts in development of dentition. The growth concepts that need to be observed during orthodontic treatment are:

- Pattern
- Variability

- Timing
- Differential growth.

The general principles of growth are outlined in Chapter 4. Hence, only the clinical significance or dynamics of growth will be dealt with in this section. *Pattern* is the physical arrangement of body at any given time. With reference to growth, pattern is changing spatial proportions over time. One of the patterns by which growth is observed in humans is the cephalocaudal gradient of growth. According to Proffit, cephalocaudal gradient means that "there is an increasing axis of growth from the head to the feet". Growth of parts closer to head grow and mature early whereas those away from the head not only complete their growth late but also grow more than the cephalic parts. Legs grow more than the trunk, but legs occupy less than 50 percent of body length at birth whereas head that occupies 25 percent of body length (1/4th) comes to occupy only 1/8th in an adult. The importance of cephalocaudal gradient of growth to us is clearly understood. Cranium is 63 percent of adult size at birth; face:cranium ratio at birth is only 1:8. So also are maxilla and mandible, maxilla completes growth early whereas mandible seems to grow more and appears to take its own time. It is not surprising to see mandibular surge in adolescents whereas maxillary growth is hardly evident after 11 to 12 years of age. This aspect of mandibular growth should be taken into account while treating patients with skeletal malocclusion with fault in mandible. Skeletal class II patients with mandibular retrognathism are treated usually with functional appliances if they are in the "growing age". This key word is not self-explanatory. We should know what the growing age of mandible really is. It is now, to an extent, understood that utilization of pubertal growth spurt in class II skeletal cases treated with functional appliances may actually cure the class II skeletal condition. On an equivalent level, when treating a skeletal class III patient due to mandibular prognathism, cephalocaudal gradient of growth makes us understand that there might be residual growth of mandible after the pubertal surge. Of all the bones in the facial skeleton, mandible is the one that has relatively higher potential to grow (partly attributed to secondary cartilage, the condyle) and the time limit for mandibular growth is not as restricted as that of maxilla or cranial base. This should be utilized in the treatment of skeletal malocclusion involving mandible.

Another aspect of pattern with equal relevance is the *Scammon's growth curve*. If cephalocaudal gradient of growth attempts to explain the quantity of growth/gradient in the amount of growth attained over time, Scammon's curve tries to provide an outlook of growth pattern of different tissues of the body. Different tissues grow at different rates and time. This is the gist of Scammon's curve. Neural tissue grows rapidly during the early stages of life and by 8 years almost 95 percent of neural growth is complete. The somatic growth follows an 'S' shaped curve, there is decreased growth rate during childhood and an increase during puberty. Maxillary and mandibular growth curves are between the neural and general tissues. Maxillary curve follows the neural growth curve closer than the mandible. It is now easy to comprehend that maxillary growth is completed quite early in life. Orthopedic appliance therapy to correct maxillary deficiency (face mask) should be started in deciduous or mixed dentition but after the eruption of all permanent teeth, there is little or no scope for correction of maxillary retrognathism. Mandibular growth follows the general body growth curve, with slowing of growth in childhood and peaking during puberty. These concepts of growth show us how different maxilla and mandible are in growth and how different they should be dealt with during treatment, though they are in absolute contact with each other (occlusion).

Timing of growth varies with individuals and also with gender. Girls complete growth earlier than boys. Pubertal spurt in growth is relevant to orthodontic treatment. Identification of pubertal spurt is not easy. Pubertal growth spurt is easily missed in early maturing girls. In late maturers, the pubertal spurt may not have started at all but the functional orthopedic treatment would have been completed. An astute clinician would know that this disproportionate growth would continue into adolescence. Hence, it is prudent to look for the starting of pubertal spurt. Skeletal and biological maturity indicators help in assessing the skeletal age of a person. The use of hand wrist radiograph to predict mandibular growth has been under scrutiny for some time. It has been questioned whether mandible undergoes spurt in growth at the same time as the other skeletal structures or it has a late surge. Now the table has shifted towards the use of hormones as an indicator of sexual maturity. Hormones are definitive indicators of growth spurts. They also help in the detection of juvenile acceleration in

growth, which is otherwise named adrenarche (Parker & Mehesh) and is due to early rise in adrenal androgens. Dehydro epiandrosterone (DHEA) and its sulphated derivatives are secreted from the adrenal cortex; they potentiate the action of growth hormone and stimulate the proliferation of cartilage cells in the epiphysis. It is found that adrenarche occurs two years prior to the start of pubertal acceleration. In fact, Sizonenko et al postulate that it is the adrenarche that stimulates the secretion of gonadotrophic releasing hormones from the hypothalamus and initiates the functioning of gonadostat (gonadotrophic axis from hypothalamus – pituitary – gonad). The maturation of gonadostat is important for the onset of pubertal spurt. Timing of class II and III treatment has been discussed exhaustively by Bacetti and McNamara with the help of various studies on treatment timing of functional appliances. Treatment of class II malocclusion with functional appliances appears to yield excellent results when it is performed at the circumpubertal period. Two studies by Bacetti et al in the treatment of skeletal class II malocclusion with Frankel and twin Block with difference in treatment time—before the onset of pubertal growth spurt and at the circumpubertal period were very conclusive in their result, favoring the treatment timing around the circumpubertal period. The results revealed that increase in mandibular length is twice as much in the circumpubertal group than in the prepubertal treatment group. Studies of McNamara, Lund and Sandler also yielded similar results. Hence, the treatment of skeletal class II malocclusion due to mandibular retrognathism is most effective when performed in the circumpubertal period. Cervical vertebrae maturation indicator to assess the skeletal maturation was used and a new index correlating it with mandibular growth was formulated by Bacetti et al. In this index, the peak of mandibular growth was found to occur within a year after attaining CS3. Functional treatment of class II at the stage CS3–CS4 yielded maximal results. This is just another indicator that mandibular growth closely follows general body growth.

Treatment of class III malocclusion should be done early. Maxillary retrognathism is not easily corrected because maxillary growth follows the neural growth curve and is completed early. In other words, circummaxillary sutures are fused during adolescence and correction of maxillary retrognathism is less effective after 10 years of age. Maxilla yields to protraction force

only if the treatment is started in mixed or deciduous dentition stage. The idea behind including an expansion screw in the maxillary orthopedic protraction appliance is not only expansion of maxilla but also loosening of circummaxillary sutures so that they respond readily to protraction force. Once the sutures are interdigitated, protraction becomes difficult and the treatment results are compromised. Mandibular prognathism treated during adolescence responds well, the reason being obvious.

Transverse maxillary correction by skeletal expansion has similar restriction as the protraction of maxilla. According to Melsen, the intermaxillary suture is smooth and open in children (6-8 years), in early adolescence (10-12 years) the sutural edges are overlapping, but in late adolescence (14-16 years) the sutures become interdigitated and fused. Bacetti summarizes Melsen's findings by quoting that maxillary expansion can be skeletally effective if the treatment is completed in early adolescence. Growth modification procedures are best done by having the timing of treatment in mind. Exact time of start of treatment varies with the gender but the results of studies shown above are a generalization of pattern that is to be followed for treating different kind of malocclusions.

All children do not have the same increments at the same time. Growth is highly variable and differential. *Variability in growth increments* is assessed by plotting the growth on a graph for a particular time frame. Growth variability curves can either be the *distance curve*, where the height of the child in centimeters is plotted at the end of every year; or the *velocity curve*, where the yearly increment in height is marked on a graph. Distance and velocity curves are used for individuals, but on a larger scale when assessing the growth of a child with respect to a population. Charts like Wetzel's grid can be used to find out whether the growth of the child follows the normative standards. These curves can help us assess the magnitude of growth of an individual child that can be correlated with the skeletal maturity indicators to ascertain the growth status.

During the pubertal spurt, another important parameter of growth that needs to be considered is the *growth direction*. There is a change in direction of mandibular growth from vertical to horizontal. Not only is the functional treatment dependent on growth direction but extraction/ non extraction decision, time

taken for extraction space closure, prognosis of other orthopedic therapy like expansion, mesial migration of teeth distal to extraction site, time taken for treatment, response to a particular treatment are all dependent on growth direction. The change from vertical to horizontal direction, if any, should be looked for.

Dimensions of the face complete growth of width first, followed by depth and height. The width of mandible is completed first, the mandibular intercanine width is established at 9 years in girls and 10 years in boys. There is hardly any increase in width after this age. The maxillary canine erupts after the mandibular intercanine width (in other words, mandibular anterior arch width) is established, infact it appears that the maxillary canines waits for the mandibular horizontal spurt, to be completed. During the pubertal growth spurt, there is a change in direction of growth of mandible from vertical to horizontal. Thus, maxillary intercanine dimension acts as a safety valve for the horizontal mandibular spurt in puberty. Width increase in maxilla is not possible after 12 to 13 years in girls, but in boys maxillary intercanine dimension increase is seen till 18 years of age. The clinical implication is that in cases of crowding, any attempt to treat by expansion would be wrought with failure due to the inability to attain a stable increase in intercanine width. There will be disturbance of equilibrium in musculature which will add to the failure. Width of the face thus follows the neural curve with depth and height following general body growth of Scammon's curve. The anteroposterior dimension of face completes next, followed by height. Downward and forward growth of maxilla is seen till 14 to 15 years in girls. Increase in height is due to separation of the jaws during displacement, growth of alveolar bone and eruption of teeth. Late increase in height of face is seen particularly in the lower third. According to Behrent, forward growth of jaws is noticeable after puberty and in adulthood and modest increase in the vertical growth of jaws is seen in adulthood.

During the development of dentition, a number of unstable occlusal states called *transient malocclusions* are encountered. To name a few, they are flush terminal plane relation, spacing in deciduous dentition—primate spaces, ugly duckling stage and these are best left untreated. Deciduous dentition presents with a transient deep bite which is corrected by contributions like change in the axial inclination of the permanent teeth when they

erupt (deciduous teeth are more upright), physiological bite raisers, etc. In certain instances, this deep bite may restrict the downward and forward growth of mandible, thus the full intercanine width might not be expressed in the mandible. The narrow mandibular intercanine width might be accentuated by the presence of class II skeletal base with increased overjet. Simple procedures like giving a bite plane might be enough to relieve the bite and facilitate forward growth of mandible.

The postnatal growth of face is not only complex but as already mentioned, the remodeling pattern of facial structures is highly specific and selective. The growth of jaws is determined by the growth of functional matrix and cranial base. Remodeling follows a specific pattern that appears to be repetitive and inherent to the human race. Balanced facial form and functions are derived from a harmonious integration of the various components of the craniofacial complex. These components grow and develop throughout life in a sequential, predictable, and orderly fashion, albeit with a wide range of variation in the amount and timing of growth. The knowledge of growth-related changes is essential in planning orthodontic treatment. It is important to understand and anticipate the amount and relative rate of growth in different parts of the face, especially during childhood and adolescence. The orthodontist needs to assess the developmental status of the individual and estimate the remaining growth to plan treatment. Diagnosis and treatment planning of an orthodontic patient must, therefore, include application of knowledge in craniofacial growth and dental development.

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Chapter

7

Development of Human Dentition, Supporting Structures and Occlusion

CHAPTER OUTLINE

- Prenatal Development of Maxilla and Mandible
 - Development of mandible
 - Development of maxilla
 - Dental arch and tooth development
 - Development of supporting structures
- Chronology of Human Dentition
- Eruption of Tooth
- Evolution of Tooth
- Development of Occlusion

As a general rule, understanding things from the beginning is most advantageous. This helps in locating the origin and magnitude of problems. Although the tooth is regarded as a separate unit or entity in biology, it is better understood in context with the total development of the tooth and the base of the jaws.

PRENATAL DEVELOPMENT OF MAXILLA AND MANDIBLE

Both the maxilla and mandible develop from the tissues of the first pharyngeal arch, the mandible forming within the mandibular process and maxilla within the maxillary process.

Development of Mandible

The cartilage of the first arch (Meckel's cartilage) has a close positional relationship to the developing mandible but makes no contribution to it (Fig. 7.1). At six weeks of development, this cartilage extends as a solid hyaline cartilaginous rod, surrounded by fibrocellular capsule, from the developing ear region to the midline of the fused mandibular processes. The two cartilages of both

the sides do not meet at the midline but are separated by a thin band of mesenchyme. On the lateral aspect of the Meckel's cartilage, during the sixth week of embryonic development, a condensation of the mesenchyme occurs in the angle formed by the division of inferior alveolar nerve into the incisive and mental branches.

At seven weeks, intramembranous ossification begins in this condensation forming the first bone of the mandible. From this center of ossification, bone formation spreads rapidly, anteriorly to the midline and backward to a point where mandibular nerve divides into lingual and inferior alveolar branches. This spread of new bone formation occurs anteriorly along the lateral aspect of Meckel's cartilage, forming a trough consisting of lateral and medial plates that unite beneath the incisive nerve. This trough of bone extends to the midline, where it comes into close approximation with a similar trough

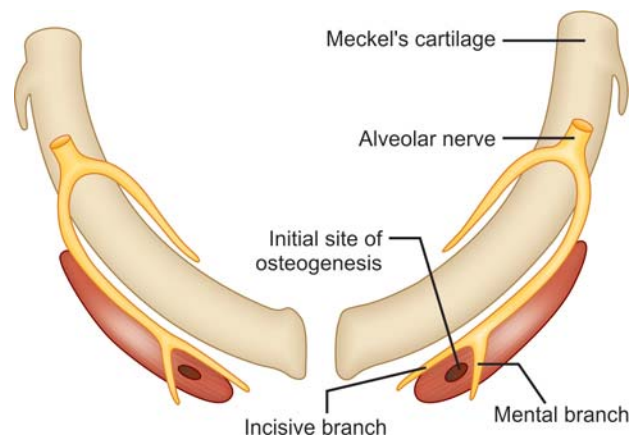


Fig. 7.1: Development of mandible. Site of initial osteogenesis related to mandible

formed on the opposing side and these two remain separate until they unite shortly after birth. The trough soon forms a canal as bone forms over the nerve. The backward extension of the ossification also forms in a similar way forming a bony canal enclosing the inferior alveolar nerve. From this bony canal, extending from the division of the mandibular nerve to the midline, medial and lateral alveolar plates of bone develop in relation to the developing tooth germs, so that the tooth germs occupy a secondary trough of bone. This secondary trough is partitioned, and the teeth come to occupy individual compartments, which finally become totally enclosed by growth of bone over the tooth germ. This is essentially how the body of mandible forms.

The ramus forms by a rapid spread of ossification backward into the mesenchyme of the first arch diverging away from the Meckel's cartilage. By tenth week of IU life, rudimentary mandible is formed entirely by membranous ossification without the involvement of Meckel's cartilage.

The condylar cartilage appears during the twelfth week of development and rapidly forms a cone or carrot shaped mass which is quickly converted into bone by endochondral ossification.

Other secondary cartilages like coronoid cartilage and symphyseal cartilage form later during development. Coronoid cartilage is a transient growth cartilage and disappears long before birth. The symphyseal cartilages, two in number, present at midline between the two mesial ends of Meckel's cartilage, are obliterated in the first year of life.

Development of Maxilla

The maxilla develops from a center of ossification in the mesenchyme of the first arch; in contrast to the mandibular process. However, the centre is in the maxillary process. No arch cartilage or primary cartilage exists, but the center of ossification is closely related to the cartilage of the nasal capsule. Center of ossification is in the angle where anterior superior alveolar nerve is given off from the inferior alveolar nerve. From here, ossification spreads forwards and backwards to form different processes of maxilla. Ossification also spreads into the palatine process to and from the hard palate. A trough of bone is formed enclosed by medial and

lateral alveolar plate which eventually forms compartments for the developing tooth germs as described in mandible.

Dental Arch and Tooth Development

Around the twenty fourth to the thirty eighth day of IU life, the epithelium covering the arches, begins to proliferate and forms an epithelial thickening on the inferior border of the maxillary process and the superior border of the mandibular arch. The thickened area of epithelium is *odontogenic epithelium*. Around the thirty seventh day, when the processes fuse on either side a single band of thickened odontogenic epithelium is formed and is known as the *primary epithelial band*. This primary epithelial band is an arch shaped continuous plate of odontogenic epithelium which forms upper and lower dental arches. These bands are roughly horse shoe shaped and correspond in position to the future dental arches in the presumptive upper and lower jaw. This band of epithelium quickly gives rise to two subdivisions—the vestibular lamina and the dental lamina. The vestibule forms as a result of the proliferation of the vestibular lamina into the ectomesenchyme. Its cells rapidly enlarge and then degenerate to form a cleft which becomes the vestibule between the cheek and the tooth bearing area. The dental lamina gives rise to tooth proper. The dental lamina connects the developing tooth bud to the epithelial layer of the mouth for a significant period of time. Within the dental lamina, continued localized proliferation leads to the formation of series of a epithelial ingrowths into the ectomesenchyme at sites corresponding to the future deciduous teeth. From this point, the development of teeth proceeds in three stages—*bud stage*, *cap stage* and *bell stage*. These terms are descriptive of the morphology of the developing tooth germ. As the development is a continuous process, clear distinction between these stages is not possible.

Tooth Development

The tooth bud (sometimes called the tooth germ) is an aggregation of cells which eventually forms the tooth. These cells are derived from the ectoderm of the first branchial arch and the ectomesenchyme of the neural crest. The tooth bud or germ (Fig. 7.2) is organized into three parts: *the enamel organ*, *the dental papilla* and *the dental follicle*.

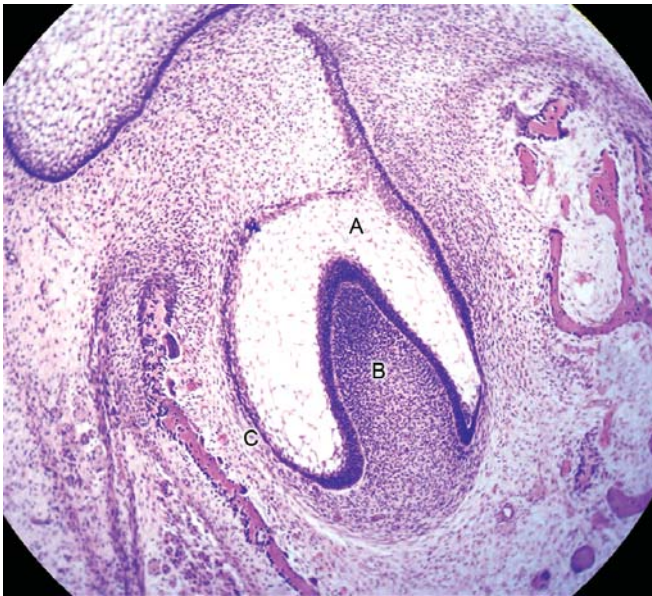


Fig. 7.2: Histologic slide showing tooth germ: (A) Enamel organ; (B) Dental papilla; (C) Dental follicle

The *enamel organ* is composed of the outer enamel epithelium, inner enamel epithelium, stellate reticulum and stratum intermedium. These cells give rise to ameloblasts, which produce enamel and the reduced enamel epithelium. The location where the outer enamel epithelium and the inner enamel epithelium join is called the cervical loop. The growth of cervical loop cells into the deeper tissues forms Hertwig's Epithelial Root Sheath, which determines the root shape of the tooth.

The *dental papilla* contains cells that develop into odontoblasts, which are dentin-forming cells. Additionally, the junction between the dental papilla and inner enamel epithelium determines the shape of the crown of the tooth. Mesenchymal cells within the dental papilla are responsible for the formation of the tooth pulp.

The *dental follicle* gives rise to three important entities: cementoblasts, osteoblasts, and fibroblasts. Cementoblasts form the cementum of the tooth. Osteoblasts give rise to the alveolar bone around the roots of the teeth. Fibroblasts develop the periodontal ligaments which connect the teeth to the alveolar bone through the cementum.

Bud Stage

The bud stage is characterized by the appearance of the tooth bud without a clear arrangement of cells. The stage

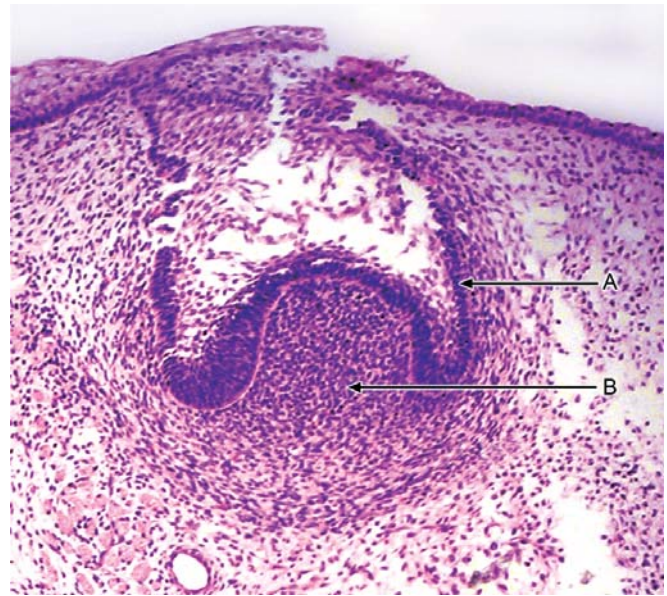


Fig. 7.3: Cap stage of tooth development. (A) Enamel organ; (B) Dental papilla

technically begins once epithelial cells proliferate into the ectomesenchyme of the jaw. The tooth bud itself comprises of the group of cells remaining at the end of the dental lamina.

Cap Stage

The first signs of an arrangement of cells in the tooth bud occur in the cap stage. A small group of ectomesenchymal cells stop producing extracellular substances, which results in the aggregation of these cells which are called the dental papilla. At this point, the tooth bud grows around the ectomesenchymal aggregation, taking on the appearance of a cap (Fig. 7.3), and becomes the enamel (or dental) organ.

A condensation of ectomesenchymal cells called the dental follicle surrounds the enamel organ and limits the dental papilla. Eventually, the enamel organ will produce enamel, the dental papilla will produce dentin and pulp, and the dental follicle will produce all the supporting structures of a tooth.

Bell Stage

The dental organ is bell-shaped during this stage, and the majority of its cells are called the *stellate reticulum* because of their star-shaped appearance. Cells on the periphery of the enamel organ separate into three

important layers. Cuboidal cells on the periphery of the dental organ are known as the *outer enamel epithelium*. The columnar cells of the enamel organ adjacent to the dental papilla are known as the *inner enamel epithelium*. The cells between the inner enamel epithelium and the stellate reticulum form a layer known as the *stratum intermedium*. The rim of the dental organ where the outer and inner enamel epithelium joins is called the *cervical loop*. In summary, the layers in order of innermost to outermost consist of dentine, enamel (formed by inner enamel epithelium, or 'ameloblasts', as they move outwards/upwards), inner enamel epithelium and stratum intermedium (specialized stratified cells that support the synthetic activity of the inner enamel epithelium). What follows is part of the initial 'enamel organ', the middle of which is made up of stellate reticulum cells. All this is encased by the outer enamel epithelium layer. During the bell stage, the dental lamina disintegrates, leaving the developing teeth completely separated from the epithelium of the oral cavity; the two will not join again until the final eruption of the tooth into the mouth.

Clone and Field Theory: The crown of the tooth, which is influenced by the shape of the internal enamel epithelium, also takes shape during this stage. Throughout the mouth, all teeth undergo this same process; it is still uncertain why teeth form various crown shapes—for instance, incisors versus canines. There are two dominant hypotheses. The *field model* proposes that there are components for each type of tooth shape found in the ectomesenchyme during tooth development. The components for particular types of teeth, such as incisors, are localized in one area and dissipate rapidly in different parts of the mouth. Thus, for example, the "incisor field" has factors that develop teeth into incisor shape, and this field is concentrated in the central incisor area, but decreases rapidly in the canine area. The other dominant hypothesis, the *clone model*, proposes that the epithelium programs a group of ectomesenchymal cells to generate teeth of particular shapes. This group of cells, called a clone, coaxes the dental lamina into tooth development, causing a tooth bud to form.

Advanced Bell Stage: Hard tissues, including enamel and dentin, develop during this stage of tooth development. This stage is also called the *crown*, or *maturation* stage by some researchers. Important cellular changes occur

at this time. In prior stages, all the inner enamel epithelium cells were dividing to increase the overall size of the tooth bud, but rapid division, called mitosis, stops during this stage at the location where the cusps of the teeth form. The first mineralized hard tissues form at this location. At the same time, the inner enamel epithelial cells change in shape from cuboidal to columnar. The nuclei of these cells move closer to the stratum intermedium and away from the dental papilla. The adjacent layer of cells in the dental papilla suddenly increases in size and differentiates into a layer of odontoblasts, which are the cells that form dentin. Odontoblasts would not form if it were not for the changes occurring in the inner enamel epithelium. As the changes in the inner enamel epithelium and the formation of odontoblasts continue from the tips of the cusps, the odontoblasts secrete a substance, an organic matrix, into their immediate surrounding. The organic matrix contains the material needed for dentin formation. As odontoblasts deposit organic matrix, they migrate towards the center of the dental papilla. Thus, unlike enamel, dentin starts forming in the surface closest to the outside of the tooth and proceeds inward. Cytoplasmic extensions are left behind as the odontoblasts move inward. The unique, tubular microscopic appearance of dentin is a result of the formation of dentin around these extensions.

After dentin formation begins, the cells of the inner enamel epithelium secrete an organic matrix against the dentin. This matrix immediately mineralizes and becomes the tooth's enamel. Outside the dentin are ameloblasts, which are cells that continue the process of enamel formation; therefore, enamel formation moves outwards, adding new material to the outer surface of the developing tooth.

Amelogenesis

Enamel formation is called amelogenesis and occurs in the crown stage of tooth development. "Reciprocal induction" governs the relationship between the formation of dentin and enamel; dentin formation must always occur before enamel formation. Generally, enamel formation occurs in two stages: the *secretory* and the *maturation* stages. Proteins and an organic matrix form partially mineralized enamel in the secretory stage; the maturation stage completes enamel mineralization. In the secretory stage, ameloblasts release enamel

proteins that contribute to the enamel matrix, which is then partially mineralized by the enzyme alkaline phosphatase. The appearance of this mineralized tissue, which occurs usually around the third or fourth month of pregnancy, marks the first appearance of enamel in the body. Ameloblasts deposit enamel at the location of the future cusps of the teeth, alongside the dentin. Enamel formation then continues outward, away from the center of the tooth.

In the maturation stage, the ameloblasts transport some of the substances used in enamel formation out of the enamel. Thus, the function of ameloblasts changes from enamel production, as occurs in the secretory stage, to transportation of substances. Most of the materials transported by ameloblasts in this stage are proteins used to complete mineralization. The important proteins involved are amelogenins, ameloblastins, enamelines, and tuftelins. By the end of this stage, the enamel completes its mineralization.

Dentinogenesis

Dentin formation, known as dentinogenesis, is the first identifiable feature in the crown stage of tooth development. The formation of dentin must always occur before the formation of enamel. Odontoblasts, the dentin-forming cells, differentiate from cells of the dental papilla. They begin secreting an organic matrix around the area directly adjacent to the inner enamel epithelium, closest to the area of the future cusp of a tooth. The organic matrix contains collagen fibers. The odontoblasts begin to move towards the center of the tooth, forming an extension called the odontoblast process. The odontoblast process causes the secretion of hydroxyapatite crystals and mineralization of the matrix.

Root Formation

The epithelial cells of the external and internal enamel epithelium from the cervical loop proliferate to form a double layered Hertwig's epithelial root sheath. This sheath of epithelial cells grows around the dental papilla between the dental papilla and follicle, until it encloses all but the basal portion of the papilla. A thin rim of root sheath, the *epithelial diaphragm*, encloses the primary apical foramen. As the inner enamel epithelial cells of the root sheath progressively enclose more and more of the expanding dental papilla, they initiate the

differentiation of odontoblasts from the cells at the periphery of the dental papilla. These cells eventually form the dentin of the root. In this way, a single rooted tooth is formed. Multi rooted teeth form essentially in the same way, except that the primary apical foramen is divided into two or three apical foramen by tongues of epithelium growing towards each other hence dividing the single foramen.

Once the root sheath forms, it rapidly initiates root formation and then fragments. With the onset of root formation, the crown of the tooth grows away from the bony base of the crypt and it hence gets stretched. Although active mitosis takes place, the continuous stretching of the root sheath causes it to fragment to a fenestrated network around the tooth and is seen as clusters of epithelial cells called *cell rests of Malassez*.

Periodontium

The periodontium, which is the supporting structure of the tooth, consists of the cementum, periodontal ligaments, gingiva, and the alveolar bone. Cementum is the only one of these that is a part of the tooth. Alveolar bone surrounds the roots of teeth to provide support and creates what is commonly called a "socket". The periodontal ligament connects the alveolar bone to the cementum, and the gingiva is the surrounding tissue visible in the mouth (Figs 7.4 and 7.6).

Formation of the Periodontal Ligament

Cells from the dental follicle give rise to the periodontal ligament (PDL). Formation of the periodontal ligament begins shortly after the root formation begins from the fibroblasts of the dental follicle (Fig. 7.7). At an early stage of formation, the cells already have an oblique orientation and the fiber bundles they form also assume a similar orientation. These fibroblasts secrete collagen, which interacts with fibers on the surfaces of adjacent bone and cementum. This interaction leads to an attachment that develops as the tooth erupts into the mouth. The occlusion continually affects the formation of periodontal ligament. This perpetual creation of periodontal ligament leads to the formation of groups of fibers in different orientations, such as the horizontal and oblique fibers. Also, before the tooth erupts, the crest of the alveolar bone is above the cemento-enamel junction and the developing fiber bundles of the

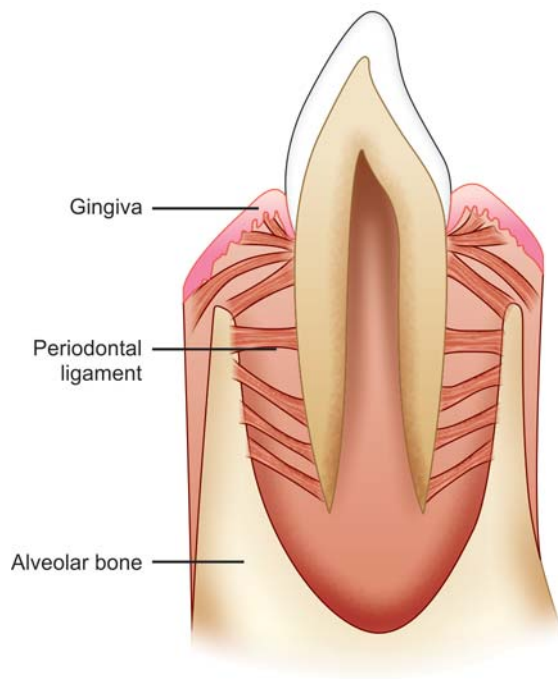


Fig. 7.4: Supporting structures of tooth

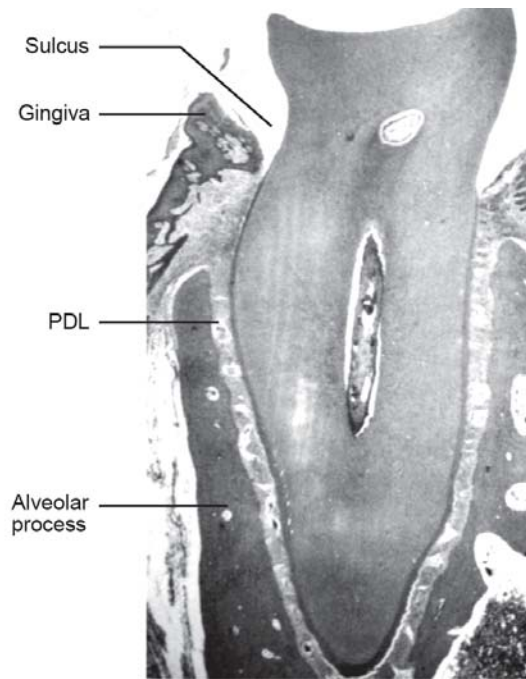
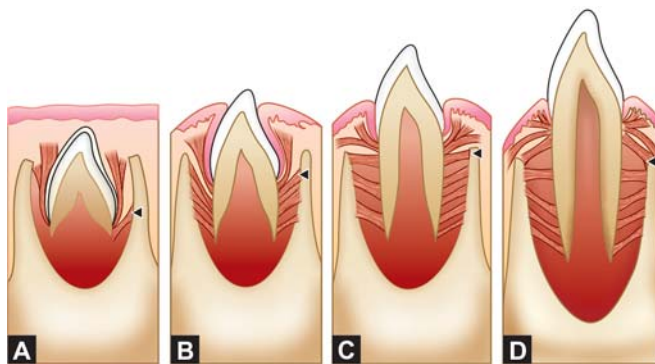


Fig. 7.6: Histological section showing periodontal ligament, alveolar bone and gingiva



Figs 7.5A to D: The development of principal fibers of periodontal ligament. The group of alveolar crest fibres (arrowheads), first forming in (A) are initially oblique (B) then horizontal in (C) and then oblique again in (D)

periodontal ligament are all directed obliquely. Because the tooth moves during eruption, the level of alveolar crest comes to coincide with the CEJ and oblique fibers become horizontally aligned. When the tooth finally comes into function, the alveolar crest is positioned near the apex.

The alveolar crest fibers have now become oblique again with a reversal in direction (Figs 7.5A to D).

Cementum Formation

Cementum formation is called cementogenesis and occurs late in the development of teeth. Cementoblasts are the cells responsible for cementogenesis. Two types of cementum form: cellular and acellular. Acellular cementum forms first. The cementoblasts differentiate from follicular cells, which can reach the surface of the tooth's root. Hertwig's Epithelial Root Sheath only after (HERS) has begun to fragment (Fig. 7.7). Once in contact with the HERS, they become large and assume all the characteristics of a protein secreting cell and secrete fine collagen fibrils along the root surface at right angles before migrating away from the tooth. As the cementoblasts move, more collagen is deposited to lengthen and thicken the bundles of fibers. Noncollagenous proteins, such as bone sialoprotein and osteocalcin, are also secreted. As mineralization takes place, the cementoblasts move away from the cementum, and the fibers left along the surface eventually join the forming periodontal ligaments. Mineralization of the organic matrix occurs with deposition of hydroxyapatite crystals and this process is slow. Due to this slow process, the cementoblasts which form the cementum retreat into the ligament and hence cementum is acellular.

Cellular cementum develops after most of the tooth formation is complete and after the tooth occludes (in contact) with a tooth in the opposite arch. The cementoblasts forming cellular cementum become trapped in the cementum they produce.

Alveolar Bone

Alveolar process is that bone of the jaws which contains the sockets or alveoli for the teeth. As root and cementum formation begin, bone is created in the adjacent area. Throughout the body, cells that form bone are called *osteoblasts*. In the case of alveolar bone, these osteoblast cells (Fig. 7.7) form from the dental follicle. The new bone is deposited around the developing ligament fiber bundles against the crypt wall. The deposition of this bone gradually reduces the space between the crypt wall and tooth to the dimensions of the periodontal ligament. Similar to the formation of primary cementum, collagen fibers are created on the surface nearest the tooth, and they remain there until attaching to periodontal ligaments.

Like any other bone in the human body, alveolar bone is modified throughout life. Osteoblasts create bone and osteoclasts destroy it, especially if force is placed on a tooth. As is the case when movement of teeth is attempted through orthodontics, an area of bone under compressive force from a tooth moving toward it has a high osteoclast level, resulting in bone resorption. An

area of bone, receiving tension from periodontal ligaments attached to a tooth moving away from it, has a high number of osteoblasts, resulting in bone formation.

Gingiva

The connection between the gingiva and the tooth is called the dentogingival junction. Information about formation of gingiva is not fully understood, but it is known that hemidesmosomes (attachment between epithelium and tooth) form between the gingival epithelium and the tooth and are responsible for the primary epithelial attachment. Hemidesmosomes provide anchorage between cells through small filament-like structures provided by the remnants of ameloblasts, the reduced enamel epithelium. Once this occurs, junctional epithelium forms from reduced enamel epithelium (one of the products of the enamel organ) and divides rapidly. This results in the perpetually increasing size of the junctional epithelial layer and the isolation of the remnants of ameloblasts from any source of nutrition. As the ameloblasts degenerate, a gingival sulcus is created.

Nerve and Vascular Formation

Frequently, nerves and blood vessels run parallel to each other in the body, and the formation of both usually takes place simultaneously and in a similar fashion. However, this is not the case for nerves and blood vessels around the tooth, because of different rates of development.

Nerve formation: Nerve fibers start to approach the tooth during the cap stage of tooth development and grow towards the dental follicle. Once there, the nerves develop around the tooth bud and enter the dental papilla when dentin formation has begun. Nerves never proliferate into the enamel organ.

Vascular formation: Blood vessels grow in the dental follicle and enter the dental papilla in the cap stage. Groups of blood vessels form at the entrance of the dental papilla. The number of blood vessels reaches a maximum at the beginning of the crown stage, and the dental papilla eventually forms in the pulp of the tooth. Throughout life, the amount of pulpal tissue in a tooth decreases, which means that the blood supply to the tooth decreases

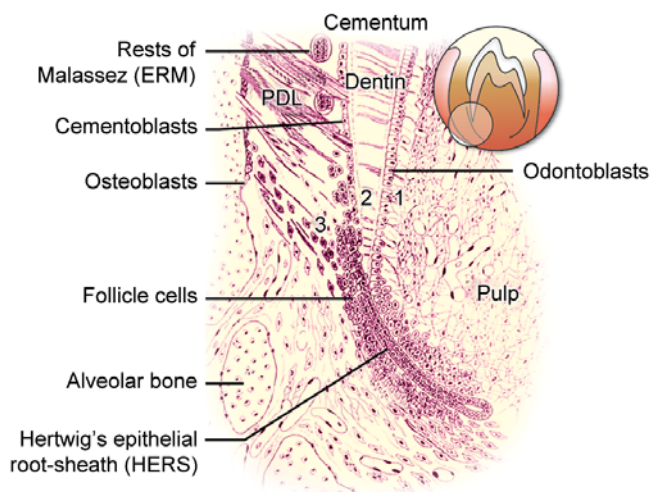


Fig. 7.7: Diagram depicting formation of tooth supporting structures: (1) Odontoblasts; (2) Fragments of HERS; (3) Osteoblasts

with age. The enamel organ is devoid of blood vessels because of its epithelial origin, and the mineralized tissues of enamel and dentin do not need nutrients from the blood.

An analysis of the successive stages of growth of the tooth germ can also be organized and studied under the following headings:

- Initiation
- Proliferation
- Histodifferentiation
- Morphodifferentiation
- Apposition
- Calcification.

Initiation: The initiation stage is first observed in the six week old fetus. This stage is recognized by the initial expansion of the basal layer of the oral cavity immediately above the basement membrane (Fig. 7.8).

Proliferation: This is actually a further multiplication of the cells of the initiation stage and an expansion of the tooth bud which results in the formation of the tooth germ in the form of a cap (Fig. 7.9). Any problem in the first two stages leads to anodontia.

Histodifferentiation: This stage is marked by the histological difference in the appearance of the cells of the tooth germ as they now begin to specialize. The tooth germ assumes the shape of bell in this stage.

Morphodifferentiation: As the name suggests, it is the stage at which the cells find an arrangement that ultimately dictates the final size and shape of the tooth. Problems in this stage leads to abnormalities in size and shape of tooth.

Apposition: The appositional stage occurs when the network or tissue matrix of the tooth is formed. This accounts for the layered appearance of enamel and dentin.

Calcification: Calcification occurs with an influx of mineral salts within the previously developed tissue matrix. Chemical structure of enamel and dentin consists of both organic and inorganic material and water. Calcification is a very slow and sensitive process. Hence, irregularities noted in a fully developed tooth could be due to systemic disturbances.

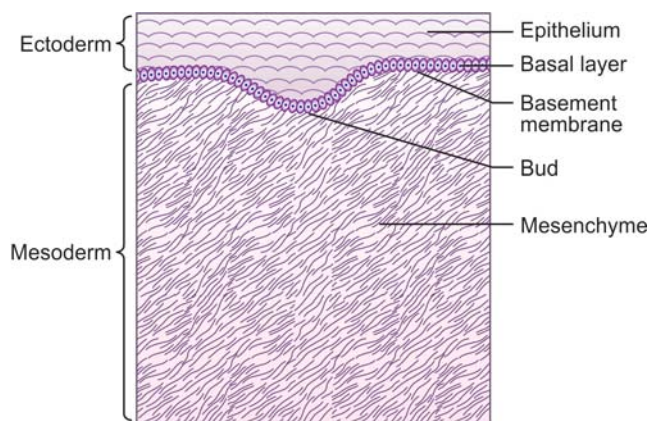


Fig. 7.8: Diagrammatic representation of initiation stage of tooth formation

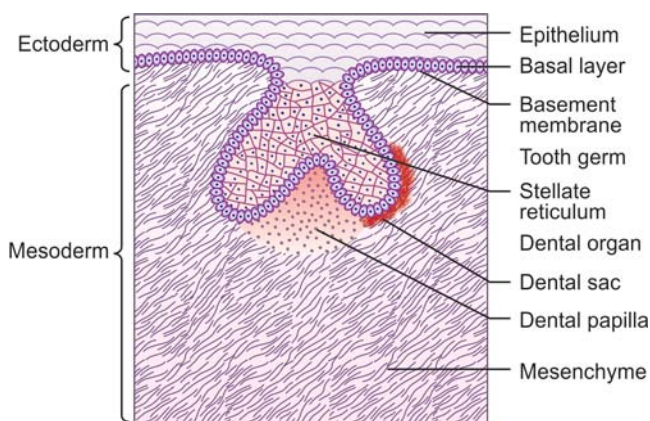


Fig. 7.9: Diagrammatic representation of proliferation stage of tooth formation

CHRONOLOGY OF HUMAN DENTITION

By determining accurately the stages of tooth development, the age of the subject under consideration can be identified. Though many earlier methods by Logan and Kronfieldb, Schour and Massler were followed, the most accurate, widely used and practically usable method was given by Nolla in 1952. In order to obtain an appraisal of the development of the particular tooth, the radiograph is matched with the figure given below (Fig. 7.10). For example, if one third of the crown is completed, the observation is given the value of 3.0; if one third of the root is completed, the observation

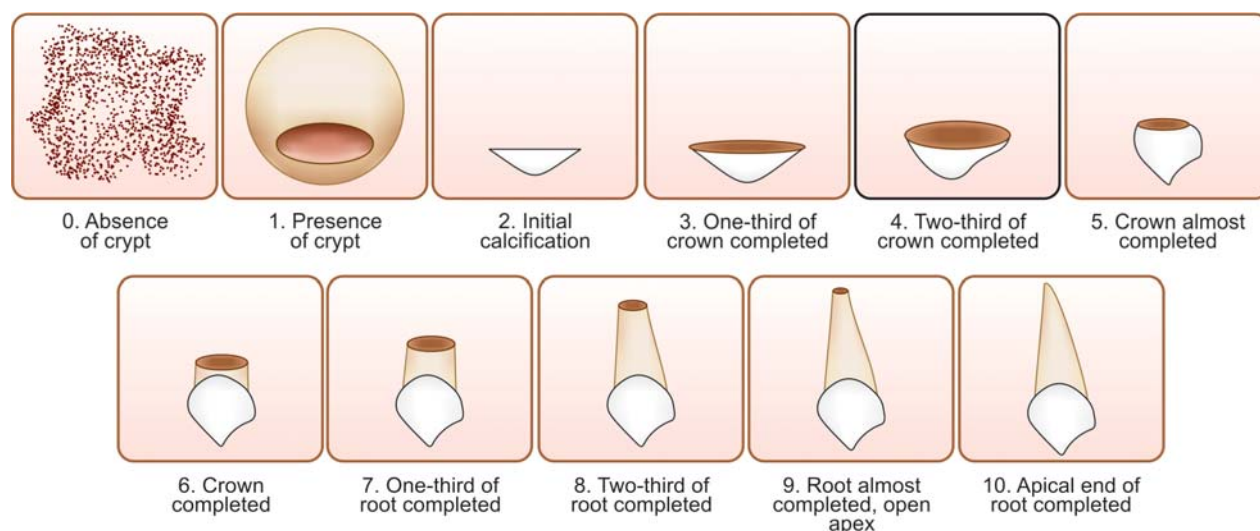


Fig. 7.10: Schematic chart indicating the ten stages of tooth development as defined by Nolla

is graded 7.0. When the radiographic reading lies between two grades, this appraisal is indicated by the value of 0.5. For example, if the X-ray reading is between one third and two-third of the root completed, it is given a value of 7.5. When the X-ray reading is slightly greater than the illustrated grade but not as much as halfway between that stage and the next, then a value 0.2 is added. For example, if it is slightly more than two thirds the crown is completed, the grade will be 4.2. If the development is slightly less than the grade indicated, the value 0.7 is added to the previous grade.

Thus, the maturation of the tooth can be used as a criterion for dental age as well.

Tooth eruptive movements begin during the sixth stage, when the crown formation is complete. After 2 to 3 years, with 2/3rd of the root formation complete, the tooth erupts into the oral cavity.

ERUPTION OF TOOTH (FIGS 7.11A TO E)

Tooth eruption is a complex series of events occurring in a continuous process to move the teeth in a three dimensional space. For teeth to become functional, considerable movement is required to bring them into occlusal plane. So tooth eruption is a developmental process and can be defined as axial or occlusal movement of the tooth from its developmental position within the alveolar crypt in the jaw to its functional position in the occlusal plane within the oral cavity.

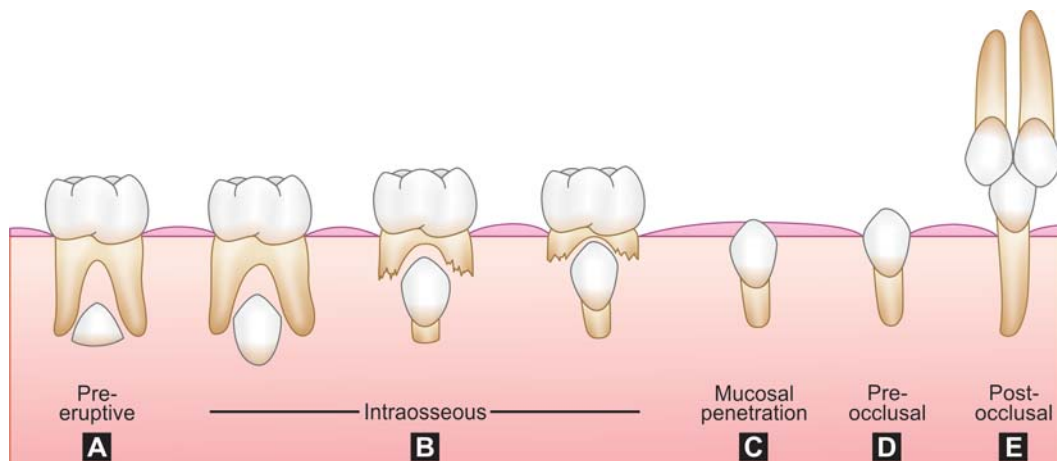
The entire process of tooth eruption may generally be described as follows:

Pre-eruptive tooth movements: Movements made by the deciduous and permanent tooth germs within the tissues of the jaw before they begin to erupt. It is that period during which the tooth root begins its formation and begins to move toward the surface of the oral cavity from its bony vault. A classical example is that of developing molars. The permanent molar tooth develops as a backward extension of the dental lamina. Initially, there is insufficient space to accommodate these new tooth germs. As a result, due to cramping of space, the upper permanent first molars develop first with their occlusal surfaces facing distally and later swing into the correct position to erupt when jaws have grown sufficiently to provide room.

Eruptive tooth movement: Made by the tooth to move from its position within the bone of the jaw to its functional position in occlusion. This phase is sometimes divided into intraosseous and extraosseous components. It is also known as prefunctional phase of tooth eruption.

Posteruptive tooth movements: This occurs after the tooth has reached its functional position in occlusion. They may be:

- Movements made to compensate for the continuous occlusal wear.
- Movements made to compensate interproximal wear.
- Movements to accommodate growing jaws.



Figs 7.11A to E: Stages of eruption: Mucosal penetration and pre-occlusal includes extra-osseous eruptive tooth movements

Mechanisms of Tooth Eruption

Pre-eruptive tooth movements are basically a combination of two factors:

Total *bodily movement* of the tooth germ and *eccentric growth* of the tooth germ where one part of the tooth germ remains fixed while the rest continues to grow, leading to change in the center of the tooth germ. These occur in an intraosseous location mainly by the bony remodeling of the crypt wall. During bodily movement, bone resorption occurs on the side of movement while apposition occurs on the opposite side. On the other hand, during eccentric tooth movements, only bone resorption occurs altering the shape of the crypt to accommodate the altering shape of the tooth germ. Whether remodeling of bone is the causative mechanism of pre-eruptive tooth movements or an adaptive response to the growing tooth germ is debatable. Pre-eruptive tooth movements consist of two important phenomena: (i) Resorption of the overlying bone and roots of deciduous tooth in the case of permanent tooth eruption and (ii) Guidance of the erupting tooth into the path created by the resorption process. Usually, both go hand in hand and resorption is the rate limiting factor in eruption process.

Eruptive tooth movements are not fully understood and most reviews on this subject have concluded that this is a multifactorial process in which cause and effect are difficult to separate. Numerous theories of tooth eruption have been proposed and there is little agreement among these theories on the identity of

mechanisms that control eruption. There have been many theories over time that have involved almost all the tissues in and around the erupting teeth and eventually all have been disproved. None of these theories alone can account for all the movements made by the tooth during its lifetime.

- **Vascular pressure and blood vessel thrust:** It is known that the teeth move in their sockets in synchrony with the arterial pulse, so local volume changes can produce limited tooth movement. Furthermore, spontaneous changes in blood pressure have been shown to influence eruptive behavior. Ground substance can swell from 30 percent to 50 percent by retaining additional water, so this also could create pressure. But experimental surgical excision of the growing root and associated tissues which eliminates the periapical vasculature did not prevent or stop eruption; this means that the local vessels are not absolutely necessary for tooth eruption.
- **Pulpal pressure and pulpal growth:** This theory says that the force exerted by the growth of cells is the result of multiplication of cells—analogue to the roots of a growing plant forcing pebbles aside. Yet, when a developing tooth is surgically removed and replaced by a silicone replica, that replica will erupt provided that the dental follicle is retained.
- **Root formation and elongation:** Root formation would appear to be the most obvious cause of tooth eruption since it undoubtedly causes an overall increase in length of the tooth along with the crown moving occlusally. Yet clinical observations,

experimental studies and histologic analyses argue strongly against such a conclusion. Rootless teeth have been found to erupt. This is most obvious in cases of dentin dysplasia type 1 and following irradiation. Experiments also indicate that some teeth erupt a greater distance than the total length of the root and teeth even erupt after root formation is completely over. Also, when a continuously erupting tooth is prevented from erupting by pinning it on to the bone, the root growth continues and is accommodated by resorption of bone at the base of socket and the bending of the root tip rather than by the occlusal movement. It is seen that, the force that normal root formation produces is sufficient to cause bone resorption and this force cannot be translated into eruptive tooth movement unless some structure exists at the base of the socket capable of withstanding the force; no such structure exists at the base of the socket. Advocates of this theory of tooth eruption supported the existence of a structure called cushion hammock ligament at the base of the socket.

- *Cushion-hammock theory:* This theory was first proposed by Harry Sicher and was widely taught between 1930's to 1950's. This theory postulated that a ligament below the tooth, which Sicher observed under the microscope, was responsible for eruption. Later the ligament Sicher observed was found to be merely an artifact created in the process of preparing the slide.

All these conclude that root formation and elongation is accommodated during tooth eruption and is a consequence and not cause of eruption process.

- *Bone remodeling:* The strongest evidence that bone remodeling as a cause of eruptive tooth movement comes from the experimental findings that even when the developing premolar is removed leaving behind the surrounding dental follicle intact, or if the developing tooth is prevented to erupt by wiring it to the lower border of mandible, an eruptive pathway still forms within the bone overlying the enucleated tooth provided the dental follicle is intact. The demonstration of an eruptive pathway cannot conclude that bone remodeling is responsible for the eruption of tooth, unless coincidental bone deposition is also demonstrated at the base of the crypt and the prevention of any such bone deposition can be

shown to interfere with tooth eruption. In human beings, the base of the crypt of permanent first molars and third molars continually resorbs as these teeth erupt while second premolar crypts show some deposition at the base. These findings, hence, preclude the notion that bone remodeling at the base of crypt causes axial tooth movement. Such experiments also indicate that dental follicle and not bone is the major determinant in tooth eruption.

The requirement of alveolar bone resorption for tooth eruption was first noted in osteopetrotic rodents. Osteopetrosis, a congenital bone disease marked by reduced bone resorption but not reduced bone formation, is often characterized by failure of teeth to erupt. For example, in the toothless rat first described by Cotton and Gaines in 1974, the teeth were fully formed but did not erupt. Such animals have fewer osteoclasts that are probably non-functional, given their weak staining for tartrate-resistant acid phosphatase. This was confirmed by scanning electron microscopy which showed the absence of bone resorption in the crypts of toothless rats, in contrast to the scalloped crypt surfaces reflecting bone resorption in normal rats.

- *Periodontal ligament traction theory:* There is a good deal of evidence that the eruptive force resides in the dental follicle—periodontal ligament complex. The periodontal ligament fibroblasts have the ability to contract and transmit the contractile force to the extracellular environment and in particular the collagen fiber bundles. The collagen fiber bundles are aligned at the correct inclination to one another to bring about the eruptive movement. This angulation of the ligament fiber bundles is a prerequisite for the tooth movement and the orientation is believed to be established by the developing root. But cases do occur where periodontal ligament is present and teeth do not erupt and cases occur in which rootless teeth erupt.
- *Dental follicle:* Originating from cranial neural crest mesenchyme, the dental follicle (DF) is a loose connective tissue sac surrounding the enamel organ of each tooth. Destined to develop into the periodontal ligament (PDL), the DF is required for eruption to occur, as not only a follicle but also perhaps as the PDL. The follicle, before it becomes the periodontal ligament, also plays a role in tooth

eruption even though it might not provide the actual eruptive force. As already discussed, if tooth germs are removed and the follicle is left intact, the eruptive pathway still forms in bone. Similarly, if a tooth is enucleated and substituted with a silicone replica within the follicle, the replica erupts, which again establishes the absolute requirement for a follicle—ligament complex to achieve tooth movement.

The requirement for the presence of the DF, or its successor, is also seen clinically. A rare disease of multiple calcifying hyperplastic dental follicles (MCHDF) is characterized by unerupted teeth with atypical follicles containing hyperplastic dense fibrous connective tissue and numerous deposits of calcified tissue. In a genetic disorder, mucopolysaccharidosis VI (Maroteaux-Lamy Syndrome), eruption of the permanent molar teeth is retarded, and the dental follicles of such teeth are abnormal in that they have excessive accumulations of dermatan sulfate. Thus, in both of these syndromes, abnormal DFs result in unerupted teeth.

- *Alveolar bone growth:* This theory was suggested by Brash based on a series of experiments using madder-fed pigs. Although bone growth is involved in tooth eruption, the cause and effect are still at the phenomenology stage.
- *Genetic input:* If tooth eruption is to be explained at the cellular and molecular level, a degree of genetic control is highly likely. In a number of genetic disorders, tooth eruption is altered.

Posteruptive Tooth Movements

Postemergent eruption consists of three stages:

Postemergent spurt: This is the phase where there is rapid tooth movement after the tooth penetrates the gingiva till it reaches the occlusal level.

Juvenile occlusal equilibrium: This is a slow process, during which teeth erupt to compensate for the vertical growth of the mandibular ramus.

When the mandible grows vertically, it moves away from maxilla creating space into which the teeth grow.

Significance of juvenile occlusal equilibrium is best understood when a tooth is ankylosed.

Adult occlusal equilibrium: This is the final phase of tooth eruption. It occurs after the pubertal growth spurt ends.

Tooth continues to erupt when its antagonist is lost and also because of wear of the tooth structure.

For accommodation of growth of jaws: These are seen as readjustment of the position of the tooth socket histologically, achieved by the formation of new bone at the alveolar crest and on the socket floor to keep in pace with the increasing height of the jaws. Recent studies indicate that this readjustment occurs between the ages of 14 to 18 years and the apices of the teeth move 2 to 3 mm away from the inferior dental canal.

Compensation for occlusal wear: The axial movement the tooth makes to compensate for the occlusal wear is most likely achieved by the mechanism similar to the eruptive tooth movement. Notably, these axial post eruptive tooth movements are made when the apices of the permanent lower first molars are fully formed and the second premolars are almost complete, which again indicates that root growth is not a factor responsible for eruptive tooth movement and further emphasizes the role of periodontal ligament.

Accommodation of interproximal wear: Wear also occurs at the contact points between teeth on their proximal surfaces. This interproximal wear is compensated by a process called the *mesial* or *approximal drift*. Three factors cause mesial drift which include the anterior component of occlusal force, contraction of trans-septal fibers between the teeth and soft tissue pressure.

When the teeth are brought into contact, an anteriorly directed force is generated. This anterior force is the result of mesial inclination of most of the teeth and summation of intercusp planes, producing a force which is directed towards causing mesial drift. The periodontal ligament plays an important role in maintaining the tooth position. The trans-septal fibers running across the alveolar process draw the neighboring tooth together and maintain them in contact. The relapse of orthodontically moved teeth is reduced if a gingivectomy removing the transseptal ligament is performed. The pressures generated by the cheeks and tongue may also push the teeth mesially.

Active tooth eruption begins in an intraosseous environment. Bone resorption, necessary for eruption, is regulated by the dental follicle. Like bone resorption, alveolar bone formation associated with tooth eruption depends upon the dental follicle and is associated with high cell proliferation. The basic principles of tooth eruption can be summarized as follows:

- Any region of a dental follicle has the potential for initiating and regulating bone resorption and bone formation.

- Movement of teeth during eruption consists of preparing a path through bone or soft tissues and moving them along this path. There is a failure of eruption when an eruption pathway has not been formed.
- Root formation is accommodated during tooth eruption and is the consequence, not cause of the process.
- Bone formation and root formation move an erupting tooth through the oral epithelium and into its position within the dental arch at the occlusal plane. It is unlikely that the periodontal ligament contributes substantially to eruption, but may play a role later in the process. Bone formation, and possibly formation of apical cementum, maintain a slow eruptive movement throughout the life of the tooth.
- Ankylosis
- Delayed root resorption.
- Carious primary teeth:
 - Apical periodontitis/cystic transformation of non-vital primary teeth
- Impacted primary teeth.
- Arch length deficiency.
- Abnormal tooth development (defects in size, shape, structure and color)
 - Regional odontodysplasia
 - Dilacerations
 - Radiation damage
 - Segmental odontomaxillary dysplasia.
- Oral clefts.

Systemic Factors

- Nutrition
- Hormonal influence
- Cerebral palsy
- Drugs, e.g. phenytoin
- HIV infection
- Anemia
- Prematurity/low birth weight
- Long-term chemotherapy
- Genetic influence
 - Familial/inherited
- Tobacco smoke
- Idiopathic.

Local Factors

Delayed tooth eruption as reported by Tomizawa et al, tends to occur in 28 to 60 percent of white people with supernumerary teeth. The most common supernumerary teeth are the mesiodens followed by the fourth molar in the maxillary arch. Tumours in the jaw, both odontogenic, like odontomas and adenomatoid odontogenic tumours and non-odontogenic, most commonly delay the tooth eruption. Mucosal barrier has also been suggested to retard tooth eruption. Any failure of the follicle of the erupting tooth to unite with the mucosa will entail a delay in the breakdown of the mucosa and constitute a barrier in the emergence of tooth. Gingival hyperplasia resulting from various causes like hormonal, hereditary or drugs results in an abundance of acellular collagen that can be an impediment for tooth eruption. Injuries to the deciduous teeth can affect the

Factors Affecting Eruption of Teeth

It is recognized that a broad range of variation exists in the normal eruption times of deciduous and permanent teeth in different persons. A valuable modification of the usually accepted chronology of the calcification and eruption times has been given by Lunt and Law. Because of this, inherent biologic variation, which is particularly noticeable in the human beings, it is difficult to determine when the eruption dates of a person are outside the limits of normal range. Nevertheless, certain cases do occur in which eruption time is grossly beyond the extremes of normality and may be considered as a pathologic state. The factors which affect tooth eruption can be grossly divided into two major categories:

1. Local factors
2. Systemic factors

Local Factors

- Physical obstruction:
 - Supernumerary teeth
 - Tumours—odontogenic and non odontogenic
 - Mucosal barrier
 - Gingival fibromatosis/gingival hyperplasia
 - Enamel pearls.
- Injuries of deciduous teeth:
 - Premature loss of primary teeth
 - Dilacerations

underlying permanent tooth and delay its eruption. Traumatic injuries can cause dilacerations of the permanent teeth, ankylosis of the injured deciduous teeth or delayed root resorption which can lead to over-retention of the deciduous teeth. Apical periodontitis or cystic transformation of a carious deciduous tooth can delay the permanent tooth eruption. Premature loss of deciduous teeth can affect eruption in both ways. Premature eruption of permanent teeth due to early loss of deciduous teeth is seen infrequently, though the delayed eruption is a commoner occurrence. The eruption of a succedaneous tooth is often delayed after the premature loss of deciduous teeth. This can be explained by the abnormal changes that might occur in the connective tissue overlying the permanent tooth germ and the formation of thick fibrous gingiva. In a recent study by Suda et al, where the relationship between formation and eruption of maxillary teeth and the skeletal pattern was studied, a shortened palatal length was found to delay the eruption of maxillary second molar. X-ray radiation has also been shown to impair tooth eruption. Ankylosis of bone to tooth, root formation impairment, periodontal cell damage, insufficient mandibular growth due to X-ray radiation seem to be involved in delaying tooth eruption.

Systemic Factors

The effect of nutrition on calcification and eruption is less significant when it is compared with other factors because it is only at the extremes of nutritive deprivation that the effects on tooth eruption have been felt. Toverud conducted an extensive study of the eruption pattern of deciduous and permanent teeth in Norwegian children during and after the World War II. During the war, these children showed a delay in the eruption of both dentitions as well as lower weight and height values for their age. Garn et al observed a clear correlation between weight, height and emergence of the permanent teeth. Barrett and Brown reported delayed eruption of the deciduous teeth among Australian aborigines which they attributed to malnutrition. McGregor et al found that Gambian children who were tall or heavy for their age tended to have more teeth than those who were short or light in weight. Disturbance of endocrine glands usually has a profound effect on the entire body, including the dentition. In hyperthyroidism and a

syndrome called adrenogenital syndrome, in which there is hyperplasia or tumours of the adrenal gland resulting in hyper secretion of the gland, premature eruption of teeth has been observed. Hypofunctioning of endocrine glands like hypothyroidism, hypopituitarism and hypoparathyroidism are most commonly associated with delayed eruption. Seow et al clearly identified a relation between preterm—low birth weight babies and number of erupted teeth. A correlation between human immunodeficiency virus (HIV) infection and delayed tooth eruption has been suggested. A study of dental manifestations in 70 children perinatally infected with HIV indicates that delayed dental eruption was directly associated with other clinical symptoms.

Genetic disorders and tooth eruption: The common genetic disorders where the tooth eruption is affected are given in Table 7.1.

To date, of the 25 known human syndromic conditions that involve disruptions in the eruption process, approximately half have led to the identification of a causative genetic mutation. For example, defects in the TRAF-6 gene in osteopetrosis where there is inadequate resorption of hard tissue throughout the body, defect of the cathepsin K gene in pyknodysostosis and COL1A1 and COL1A2 gene in osteogenesis imperfecta have been identified. Clearly, there is considerable amount of work remaining before the genetic etiology of the remaining eruption defects can be completely determined. In most of these human conditions, however, the mode of inheritance has already been determined (divided nearly equally between autosomal-recessive and -dominant or between X-linked recessive and dominant). But, while most eruption defects are part of a genetic syndrome, they can also be non-familial (caused by sporadic mutation). Classified as the most intriguing among these conditions affecting tooth eruption is the Primary Failure of Eruption (PFE), where localized failure of eruption of permanent teeth exists with no other systemic involvement. This condition affects mainly permanent posterior teeth that are fully formed but are unable to reach the occlusal plane due to a primary defect in the eruption mechanism itself (Kaban et al, 1976; Proffit and Vig, 1981; Brady, 1990; Piattelli and Eleuterio, 1991). Teeth affected by PFE are not impacted by any structures and are not ankylosed, thus making this condition one of the most difficult to diagnose

Table 7.1: Genetic disorders of eruption

<i>Syndrome/Condition</i>	<i>Eruption Phenotype</i>
Cleidocranial dysplasia	Delayed eruption
Osteopetrosis	Failure of eruption
GAPO syndrome	Failure of eruption
Osteopathia striata with cranial sclerosis	Failure of eruption in some cases
Osteoglophonic dysplasia	Failure of eruption of 2° teeth
Singleton-Merten syndrome	Dysplastic development with delayed eruption of 2° teeth
Aarskog syndrome	Delayed eruption
Acro dysostosis	Delayed tooth eruption (23% of cases)
Albright hereditary osteodystrophy	Delayed eruption
Apert syndrome	Delayed and ectopic eruption
Chondroectodermal dysplasia (Ellis-van creveld syndrome)	Delayed eruption partial/anodontia
Cockayne syndrome	Delayed eruption
De Lange syndrome	Delayed eruption
Dubowitz syndrome	Delayed eruption and hypodontia
Frontometaphyseal dysplasia (Gorlin-Cohen syndrome)	Delayed eruption and retained deciduous teeth
Goltz syndrome (Focal dermal hypoplasia)	Delayed eruption and hypodontia with hypoplastic teeth
Hunter's syndrome	Delayed eruption
Incontinentia pigmenti	Delayed eruption, hypodontia in 80%
Killian/Teschler-Nicola syndrome	Delayed eruption
Levy-Hollister syndrome	Delayed eruption of 1° teeth
Maroteaux-Lamy Mucopolysaccharoidosis syndrome	Delayed eruption with small teeth
Osteogenesis imperfecta Syndrome Type I	Delayed eruption, dysplastic teeth
Progeria syndrome (Hutchinson-Gilford syndrome)	Delayed eruption of 1° and 2° teeth and hypodontia of 2° teeth
Pyknodysostosis	Delayed eruption and occasional anodontia
Primary failure of eruption	Failure of 2° teeth to erupt partially or completely

and treat among the human anomalies of tooth eruption. Attempts to close the resultant open bite orthodontically are futile and may result in ankylosis of PFE-affected teeth. The genetic and molecular basis of PFE is not yet known. However, unique features of PFE offer exciting possibilities for unravelling the molecular basis of eruption failure. Since PFE exclusively affects posterior teeth without the involvement of any systemic disorder, we can deduce that the candidate gene for PFE would be molecules that function solely in the pre-eruptive phase and that are expressed in cells of the dental follicle and

surrounding structures. Hence, it is likely that genes like CSF-1, NF- κ B, and c-fos are prime and equally likely candidate genes responsible for the eruption defect in human PFE. Also, tooth eruption is regulated by various cytokines including epidermal growth factor, transforming growth factor- β , interleukin-1 and colony stimulating factor. Lack of appropriate inflammatory response, an inadequate response of some cytokines and increased bone density that impedes resorption have been noted to be factors for delayed tooth eruption that are genetically controlled in some syndromes.

EVOLUTION OF TEETH

Teeth as a feeding mechanism in an oral cavity (mouth) are functionally and locationally linked with jaws. In fossils, teeth found in the oral cavity are usually linked with jaws, although mineralized structures with the same histology as teeth were seen in fossils from a period before jaws appeared. Denticles in the skin occur in both fossils and extant fish. Pharyngeal denticles also occur in both extant and fossil gnathostomes but in only a few fossil agnathans (thelodonts). Complex structures with dentine and enamel have been described in the earliest jawless vertebrates, conodonts. Such fossils have been used to suggest that teeth and jaws have evolved and developed independently. Our understanding of the developmental biology of mammalian tooth development has increased greatly in the last few years to a point where we now understand some of the basic genetic interactions controlling tooth initiation, morphogenesis and patterning.

From an evolutionary-developmental perspective, there are four important features that make teeth an attractive model system: (1) Cusp patterns, tooth shapes and their arrangement in a dental pattern are unique to each species and are as indicative of a species as its DNA. (2) Because tooth pattern is intimately linked to feeding and hence survival, changes in tooth pattern in evolution provide a major basis for adaptations linked to exploitation of new feeding niches. (3) Tooth development is a simple process, involving just two embryonic cell types. (4) Embryonic tooth primordia can be easily cultured *in vitro* to completely recapitulate normal development. This enables many different types of experimental manipulation to be carried out, including recombination between different species.

The Evolution of Teeth and Jaws—The Agnathan to Gnathostome Transition

The evolution of teeth is believed to have occurred by one of two different mechanisms: (1) Teeth evolved independently from jaws from pharyngeal denticles, similar to those found in many extant species of fish such as zebra fish (Smith & Coates, 1998, 2001); (2) Teeth evolved at the same time as, or after, jaws by internalisation of skin denticles (dermal armour) similar to those found on modern day sharks (Reif 1982, reviewed by Smith & Coates, 2001). Orofacial development in a species that have teeth, cartilage and bone involves the same genes as the development of a species with cartilage and bone but no teeth. Functional data, principally from gene targeting (knockouts) in mice, show that although there are specific genetic pathways involved in tooth and jaw development, tooth morphogenesis shares many key genes with jaw skeletal morphogenesis. The latter suggests that these two tissues evolved independently but that the evolution of heterodonty (teeth with different shapes) from homodonty (teeth with one simple, conical shape) involved co-option of existing genetic pathways controlling jaw skeletal morphogenesis. Disruptions that affect dental patterning also produce abnormal skeletal development of the jaws. There are, however, many examples of gene knockouts that affect jaw hard tissue development but where tooth development is normal. *Pitx1* is a homeobox gene expressed in jaw primordia mesenchyme from E9. *Pitx1* knockout mice have a very truncated mandible but despite this, teeth are present and appear normal (Lanctot et al, 1997, 1999). *Gooseoid* (*Gsc*) is a homeobox gene expressed in jaw primordia mesenchyme from E10.5 (Gaunt et al, 1993; Tucker et al, 1999). This indicates that there are genes required for early development of teeth but which are not involved in jaw development and similarly there are genes required for jaw skeletal development which are not involved in early tooth development. This genetic independence of tooth from jaw development suggests that they evolved independently. However, the fact that genes regulating dental patterning, i.e. the development of different shapes (types) of teeth, also regulate jaw skeletal morphogenesis implies that dental patterning, which is a later event in evolution, resulted from co-option of genes regulating jaw morphogenesis. The ability

of developing teeth to adapt genetic pathways that were in place to regulate jaw morphogenesis may have thus represented an important step in the evolution of heterodonty. In humans, there are numerous examples of tooth patterning abnormalities that occur in the absence of skeletal abnormalities. Thus, for example, hypodontia (missing teeth) can occur in the absence of any obviously abnormal jaw phenotype. Significantly, it is the replacement teeth in humans that are almost always affected in hypodontia, whereas the deciduous dentition develops normally. This suggests that there are important aspects of the development of permanent teeth that involve different genetic control to deciduous tooth development or jaw skeleton formation.

Control of Dental Patterning

Tooth shape is indelibly linked to position in the jaws. Tooth shapes have evolved for particular functions. Incisors and canines are grasping/cutting teeth, premolars and molars are both grinding and cutting teeth. In heterodont dentitions, there is little point in having grasping/cutting teeth at the rear of the mouth and grinding teeth at the front. The observations that a number of different homeobox genes are expressed in distinct spatial domains in early jaw primordia mesenchyme has led to the suggestion that determination of tooth type is regulated by these genes. The Odontogenic Homeobox Code model of tooth patterning (Fig. 7.12), that has been proposed, states that in mice, genes such as *Barx1*, *Dlx1* and *Dlx2* have specific roles in directing mesenchyme cells to follow a multicuspoid (molar) pathway of tooth morphogenesis.

Genes such as *Msx1* and members of the *Alx* family have roles in directing cells to follow a monocuspid (incisor) pathway. An additional key feature of this model is that it is not only the expression of these genes in particular mesenchymal cells that is important but also the absence of expression of other genes. Thus maxillary molar morphogenesis not only requires the presence of *Barx1*, *Dlx1* and *Dlx2* but also the absence of *Msx1* and *Alx* genes (McCollum & Sharpe, 2001). Ectopic expression of *Barx1* in distal mandibular primordia mesenchyme, accompanied by loss of *Msx1* expression results in a transformation of incisor teeth into molars. Genes that regulate molar morphogenesis also regulate proximal jaw skeletal development and genes that regulate incisor morphogenesis also regulate distal jaw

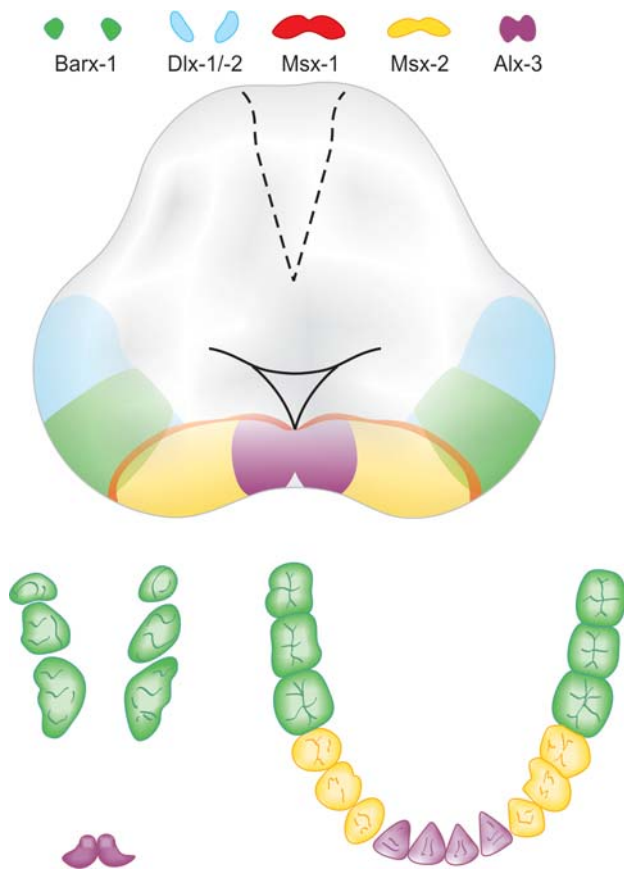


Fig. 7.12: Diagrammatic representation of the Odontogenic Homeobox Code model of dental patterning. An oral view of the mandibular arch primordium is shown with domains of several homeobox genes expressed in the mesenchyme. Expression of each homeobox gene is represented by a different color shown in the key at the top of the figure. The key also illustrates the different shaped expression domains of each of the genes in mandibular primordium ectomesenchyme

development. The indication from these data is that jaw morphogenesis and tooth patterning are controlled by the same genes.

Since, heterodont dentitions (different tooth shapes in the same dentition) evolved after homodont dentitions (teeth of all one shape), this suggests that different tooth shapes evolved by co-opting genes that were already expressed in facial primordia development to regulate jaw skeletal morphogenesis. The generation of tooth shapes other than incisors and molars is suggested to involve overlapping domains of homeobox genes. Thus, for example, the mesenchyme cells that express *Msx1*,

Dlx1 and *Dlx2* might correspond to the development of canines or premolars.

Mandible and Maxillae

Tooth patterning is very similar to patterning of the axial skeleton. Vertebral bodies are mineralized 'organs' with a basic structure that is modified according to rostro-caudal position. The morphogenesis of each individual vertebrae is fixed in any given species such that the relative order in the spine can easily be reconstructed from fossil remains. Similarly, arrangements of different shapes and sizes of teeth on the two jaws are fixed and dental patterns can be reconstructed even when the teeth are isolated from the jaws. This is particularly evident for the teeth which occlude. Mammalian molar teeth are designed to function by making specific contacts with each other on the upper and lower jaws. Molars cannot function without such occlusion. Thus, in the same way that each vertebra precisely 'fits' with immediate neighbors, each tooth aligns precisely with its counterpart in the opposing jaw. In the absence of any conflicting constraints, the most logical developmental mechanism for ensuring tooth development on opposing jaws is coordinated, would be to use the same basic genetic mechanism that is subtly modified to produce slight differences in shape between opposing teeth. It is now clear that this is not the mechanism and morphogenesis of teeth on the different facial primordia is in fact regulated by different genetic pathways. The most striking demonstration of this involves the activin signaling pathway. Activin is a member of the TGF- β superfamily of signalling proteins that binds to membrane receptors and activates gene transcription via the Smad-mediated pathway (Attisano & Wrana, 1998).

Transplantation of cells between the early mandibular and maxillary primordia has revealed that cells behave according to their donor genetic program and not as the host cells. Thus, mandibular cells that express *Dlx5* and *Dlx6* continue to express these genes when transplanted to the maxillary primordium, despite being surrounded by cells that do not express these genes. Similarly, maxillary primordium cells that do not express *Dlx5* or *Dlx6*, do not start to express these cells when transplanted to the mandible, despite being surrounded by *Dlx5* and *Dlx6* expressing cells (Ferguson et al, 2000).

Many of the genetic mechanisms that control maxillary and mandibular molar (and indeed incisor)

development are the same. It is the early responses of the mesenchyme cells to epithelial signals that establish cell position, and hence morphogenesis that are different.

The early genetic processes that influence jaw hard tissue morphogenesis thus reveal differences between the jaw primordia that are reflected in different mechanisms used to establish tooth morphogenesis. One evolutionary interpretation of this is that the different genetic pathways are related to the obviously different skeletal morphogenesis of the upper and lower jaws. Thus, for example, *Dlx5* and *Dlx6* are required for producing the normal skeletal morphogenesis of the lower jaw. Morphogenesis of teeth on the jaws involves the same genes that control skeletal morphogenesis despite the fact that the functions of these genes are unique to one or the other jaw primordium. Again, the simplest interpretation of this is that the evolution of heterodont dentitions used existing genetic pathways that were already in place to regulate jaw morphogenesis. How, therefore do these developmental data relate to the most recent evolutionary suggestions that teeth evolved before jaws? First, it is important to realize that in this view of tooth evolution, the first teeth to evolve were not oral (marginal) teeth but were embedded in the pharynx. Thus the 'teeth before jaw' hypothesis does not refer to oral teeth, as we know them in mammals. This being the case, it is not difficult to reconcile the developmental data where pharyngeal teeth moved forwards towards the oral cavity at the time of or after the agnathan to gnathostome transition. Since the genetic pathways regulating jaw morphogenesis were already in place to produce the required skeletal morphogenesis of the jaws, the pharyngeal teeth developing in the oral cavity were exposed to this information which was deployed to produce different shapes of teeth, and thus heterodonty evolved. Perhaps significantly, the differences in tooth shapes vary greatly along the anteroposterior axis (incisor-molar) in heterodonts but far less so at equivalent positions on the upper and lower jaws. This is consistent with the concept of the agnathan to gnathostome transition involving the modification of anterior arches of the segmented pharyngeal skeleton into dorsal and ventral articulating portions that were initially morphologically similar. This was then followed by considerable elaboration of these dorsal and ventral jaws during gnathostome evolution, such that the anterior

and posterior regions of each jaw evolved very different skeletal morphologies.

Theories of Mammalian Tooth Formation

The transition of mammalian teeth from other species is the subject of the theories of mammalian tooth formation. The *conrescence theory* assumes that the mammalian teeth originated by fusion of the anlagen of originally discrete reptile teeth. In the usually strong heterodont dentition of the mammals, only the incisors and canine teeth are generally simply formed. The molars and premolars assume complicated forms. According to the *conrescence theory*, a multicuspid molar is formed due to fusion of number of simple conical teeth to form an entity of higher order. It is thereby assumed that each cusp represented originally a separate tooth. Louis Bolk modified the *conrescence theory* and called it as *concentration theory*. The *concentration theory* also assumes that mammalian molar originated from several structures that are similar to tooth, but the fusion takes place in the labiolingual direction and not in fusion of tooth anlagen one behind the other. The *differentiation theory* maintains that even the most complicated mammalian molar originates from a uniform tooth anlage.

DEVELOPMENT OF OCCLUSION

In dentistry, occlusion usually means the contact relationship in function. Concepts of occlusion vary with almost every speciality of dentistry. Salzmann has defined occlusion in orthodontics as "the changing inter-relationship of the opposing surfaces of the maxillary and mandibular teeth which occurs during movements of the mandible and terminal full contact of the maxillary and mandibular dental arches". Occlusion is the sum total of many factors like genetic factors, environmental factors, and muscular pressures and occlusion constantly, changes with development, maturity, and aging.

Periods of Dental Development (Stages of occlusal development):

The development of occlusion and teeth formation can be studied under the following headings:

- Predental stage or mouth of neonate (0–6 months)
- Deciduous dentition stage (6 months–6 years)

- Mixed dentition stage (6–12 years)
 - First transitional stage
 - Second Intertransitional stage
 - Second transitional stage
- Permanent dentition

Mouth of the Neonate/Gum Pads/Pre-dentition Stage (0–6 Months)

The alveolar arches at the time of birth are called gum pads. Initially they are smooth and firm, but later get segmented corresponding to the sites of developing teeth. The basic form of the arches is determined in intrauterine life. Leighton has outlined the various factors that determine the size of gum pads as follows:

- The state of maturity of infant at birth.
- The size at birth as expressed by birth weight.
- Size of developing primary teeth.
- Genetic factors.

Maxillary arch is horse-shoe shaped and the gum pads extend labially and buccally beyond those in the mandible. Maxillary gum pads develop in two parts, namely labiobuccal and lingual portions. The labiobuccal portion grows fast. It is divided into ten segments by transverse grooves which correspond to the deciduous tooth sac. The groove between the canine and deciduous first molar is called *lateral sulcus*. Lingual portion of the arch remains smooth throughout. Labiobuccal and lingual are demarcated by the dental groove. The *dental groove* passes from the incisive papilla, runs laterally and joins with the gingival groove at the lateral sulcus area. From there it runs distally and buccally to the first molar crypt. Gingival groove demarcates the palate from gum pads.

- Mandibular gum pads are U shaped.
- Anteriorly the lower gum pad is everted.
- Gum pad is divided into ten segments by transverse grooves. The segments are less defined when compared to maxillary gum pad.

Relationships of the arch: Gum pads relationship is arbitrary. They do not have definite or precise jaw relationship or bite in the neonatal jaws. The upper lip appears short. Tongue is interposed between the lips. Maxillary gum pad is wider than mandibular gum pad and there is total overlapping of maxillary gum pads transversely and anteroposteriorly (Fig. 7.13). Lower lateral sulcus is distal to upper lateral sulcus. Vertical gap



Fig. 7.13: Gum pads—maxillary arch

exists in between the upper and lower lip pads in the anterior region. The gum pads grow rapidly during the first year of life, and the growth is more in the transverse direction. Length of the gum pad also increases, mostly posteriorly to accommodate the deciduous first and second molars.

Authors like Leighton have claimed that there is so much of variability in the relationship of upper and lower gum pads and that neonatal relationship cannot be used as a diagnostic criterion for predicting future occlusion in the primary dentition.

Occasionally, a child will be born with tooth present in the mouth. It is called as "natal tooth" when present at birth, "neonatal tooth" if it erupts during the first month and "pre-erupted teeth" if erupts during second or third month. Mandibular incisors are the most frequently erupted natal or neonatal teeth. Usually these teeth exhibit hypoplasia and pose problems or discomfort during nursing. They should be removed only if they are loosely attached and if they are supernumerary. If they are normal and firmly attached to the bone, they should not be extracted.

At birth, the tooth buds of all the primary teeth are present and are in different stages of development. Radiograph of the new born will show crowded lower incisors. The reason for this crowding is two fold: (i) The full mesiodistal width of the incisors is attained very early and (ii) The symphysis of mandible has not yet expanded fully for the incisors to unravel.

When a line is drawn along the occlusal surface, it passes through the condyle during this period (Fig. 7.14). The developing primary tooth buds lie almost on the same occlusally oriented plane (Fig. 7.15).

Primary Dentition Stage (6 Months to 6 Years)

Primary dentition stage can be studied under two headings. (i) Development of primary teeth and (ii) development of primary dentition occlusion.

Development of the Primary Teeth

- **Calcification:** The sequence and approximate timing of events in primary teeth is given in Table 7.2. The factors which control primary teeth calcification

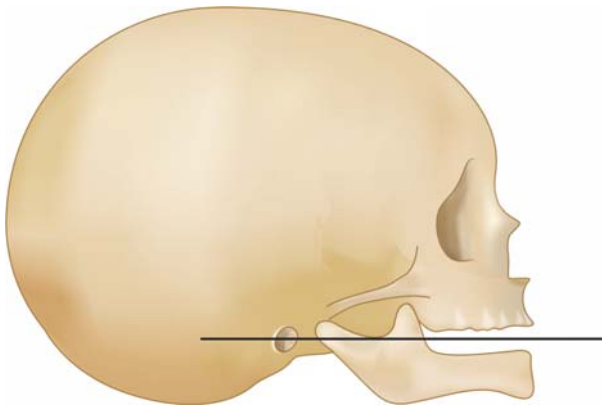


Fig. 7.14: The occlusal plane passes through the under-developed condyle in an infant

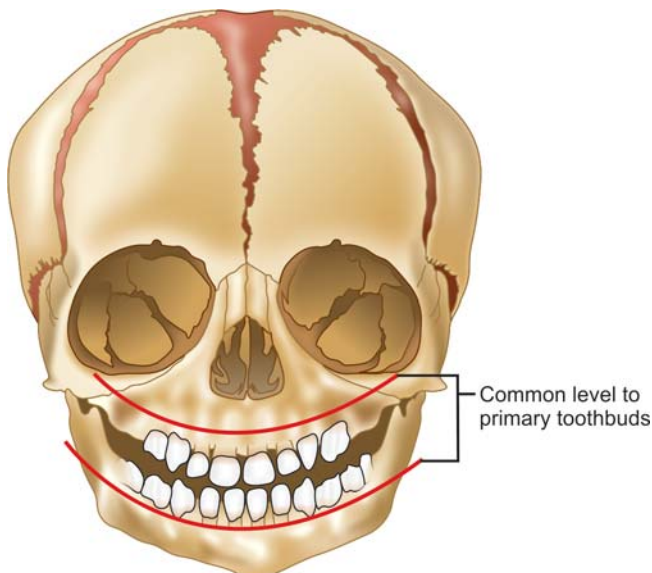


Fig. 7.15: Toothbuds position in an infant

include genetic factors, developmental variability and sexual dimorphism.

- **Eruption:** Eruption of primary tooth starts after beginning of formation of roots. The normal sequence of eruption of primary teeth is as follows: central incisors, lateral incisor, first molars, canines and second molars. Infante (1974) has shown that the emergence of tooth is highly associated with height, followed by weight and then head circumference of the individuals. The chronology of eruption of primary teeth is given in Table 7.3.
- **Features of Primary teeth:**
 - Generally, the size of primary teeth is larger in boys than those in girls.
 - Congenital missing of primary tooth is a rare occurrence. It is said to be 1 percent. The order of missing tooth is as follows: mandibular lateral incisors, maxillary lateral incisors and first molars.
 - Abnormalities in primary tooth size and shape are also few as compared to permanent teeth.
 - Eruption of permanent teeth is considered to be the sole factor in causing primary tooth resorption.

Table 7.2: Approximate timing of events in primary tooth formation

Tooth	Time of initial calcification	Crown completion in months		Root completion in years	
		Mand.	Max.	Mand.	Max.
Central incisors	14 weeks	2½	1½	1½	1½
First molar	15 ½ weeks	5½	6	2¼	2½
Lateral incisors	16 weeks	3	2½	1½	2
Canines	17 weeks	9	9	3¼	3¼
Second molar	18 weeks	10	11	3	3

Table 7.3: Chronology of primary tooth eruption

Deciduous teeth	Age of eruption
Lower central incisor	7 months
Upper central incisor	9 months
Upper and lower lateral incisors	11 months
First molars	15 months
Canines	18 months
Second molars	26 months

But primary tooth resorption is seen even in the absence of permanent successors. Factors which hasten the primary tooth resorption are occlusal trauma and inflammation, while it is delayed by the splinting and absence of permanent successor.

- The chances for ankylosis of primary teeth are greater than the permanent teeth. Mandibular teeth are more prone for ankylosis. An ankylosed tooth is often called as "submerged tooth". The exact cause for ankylosis is not clear. Ankylosis occurs during the rest periods of resorption: osseous bridging and fusion between bone and tooth occur. Ankylosis is evident during late primary and mixed dentition stages.

Development of Primary Occlusion

Features of deciduous dentition occlusion: Spaced dentition or open dentition, primary dentition in which interdental spaces are present is called Open dentition or Spaced dentition. There are two types of spacing, namely physiologic spacing (Developmental/Generalized) and Primate Space.

Developmental Spaces are present throughout the primary dentition. The reason for developmental space is anterior posterior growth of the jaws. Spaced dentition is preferable in a child because the chances for crowding in the permanent dentition are very minimal. Developmental space is on an average 4 mm in maxillary arch and 3 mm in mandibular arch.

Primate Spaces (Fig. 7.16) are present in the primary dentition in both maxillary and mandibular arches. It is also called Simian space/Anthropoid space because it is seen in monkeys. Primate space is present between deciduous lateral incisor and canine in the maxillary arch. In the mandibular arch, it is present between primary canine and primary first molar. Primate spaces are used in early mesial shift in mandibular arch.

Closed dentition/Nonspaced dentition: Primary teeth without any spaces in between teeth are called closed dentition. Lack of space could be either due to wider primary teeth or reduced arch length. Closed dentition invariably leads to crowding in the permanent dentition.

Deep bite: When the primary incisors erupt, the overbite is deep. This could be due to vertical inclination of the primary incisors. Over a period of time, this deep bite reduces due to two reasons: (1) Eruption of primary

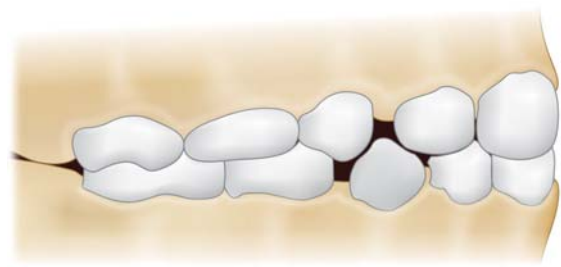


Fig. 7.16: Primate space seen in maxillary and mandibular arch

molars, (2) Rapid attrition of incisors. At about six years of age, there may be an edge to edge relationship.

Overjet: Overjet is initially more in primary dentition. The overjet decreases with the movement of the whole dental arch anteriorly. The average overjet in primary dentition is 1 to 2 mm.

Terminal plane relationships: Baume classified the relationships (Figs 7.17A to C) of the primary teeth into three categories—Straight or Flush terminal plane—seen in 76 percent; Mesial step—14 percent and Distal step—10 percent.

Baume's classification of primary teeth is based on the relationship of upper and lower primary second molar. A line is drawn along the distal surface of maxillary and mandibular second primary molar. If it is straight, it is called flush terminal plane relationship, otherwise either mesial step or distal step.

General features of deciduous dentition:

- Dental arches are normally ovoid in shape.
- Deep bite is present initially which changes to edge-to-edge relationship.
- Development spaces present.
- Flat curve of spee.
- Shallow intercuspatal contact.
- Minimal overjet.
- Straight or vertical inclination of incisors.
- Absence of crowding.

Neuromuscular considerations: Initially as the incisors erupt, interdigitation occurs in the front. Subsequent to the eruption of other teeth, the muscles learn to effect the necessary functional occlusal movements. Because of the shallow cusps of the primary teeth, there is greater adaptability of the primary occlusion. The teeth are guided into their occlusal position by the functional matrix of the muscles during active growth of the facial skeleton.

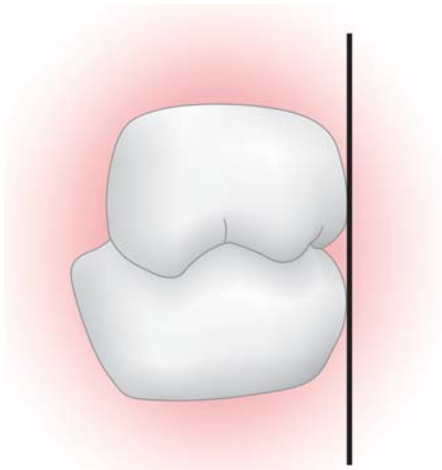


Fig. 7.17A: Flush terminal plane

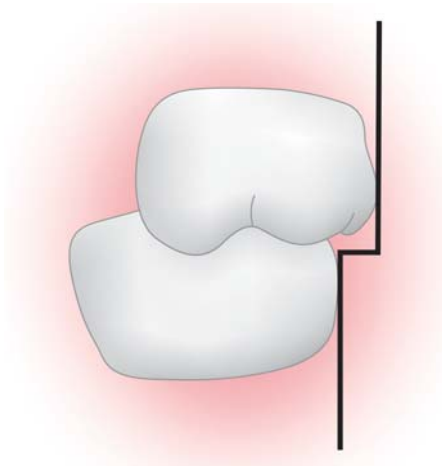


Fig. 7.17B: Mesial step relation

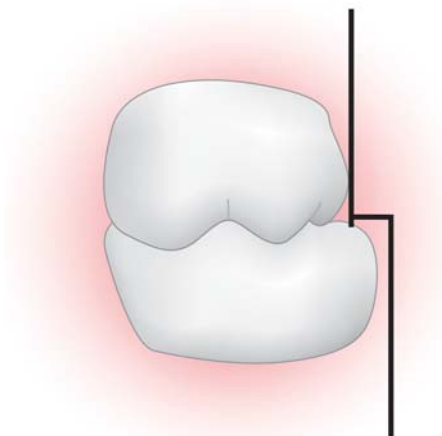


Fig. 7.17C: Distal step relation

The first three-dimensional occlusal relationship is established with the eruption of primary first molars. With the completion of eruption of primary teeth, the occlusal plane is located below the condyle (Fig. 7.18).

First Inter-transitional Period

This is the period between the completion of the primary dentition and the emergence of the first permanent tooth. During this period there is marked intrabony changes but with little changes intraorally. Vertical changes due to growth and attrition of teeth are evident. The space for the eruption of the first permanent molars is achieved by the resorption of the anterior border of ramus in mandible and apposition of bone in maxillary tuberosity. There is deepening of bite due to attrition of deciduous incisors and there is reduction in overjet also.

Mixed Dentition Stage—The Transitional Years (6-12 Years of Age)

Transition from the primary dentition to the permanent dentition begins at 6 years of age with the eruption of permanent first molars and permanent incisors. Early during this period of time, many children experience the eruption of the four permanent first molars and the exfoliation of the mandibular central incisors and the subsequent eruption of permanent incisors. It is the period during which both primary and permanent teeth are present in the mouth.

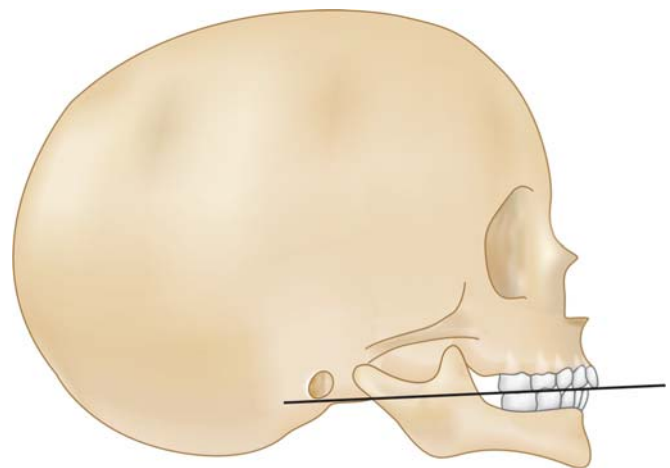


Fig. 7.18: The occlusal plane is located below the condyle in a child with complete primary dentition

This stage of occlusal development can be divided into three stages: first transitional period, second inter-transitional stage and second transitional period.

First Transitional Period

This period marks the first exchange of teeth which begins by 6 years of age and is usually complete within two years. Two important events take place in this period, namely the eruption of permanent first molars and the replacement of incisors.

Eruption of first permanent molar: The upper and lower first molars display different pathways of eruption. The lower molar buds are mesially and lingually inclined. This position is essential for development to occur in the curved junction of the ramus and alveolar process. Consequently, the lower first molar erupts in a mesial and lingual arc. The upper first molar bud develops with a buccal and distal orientation and therefore erupts in distal and buccal arc. In patients with spaced primary dentition, and flush terminal plane relationship, when the permanent mandibular first molars emerge or erupt at about 6 years, they close the primate space distal to canine. Thereby, the flush terminal plane gets converted into mesial step. This allows the permanent maxillary first molars to erupt into a Class I Molar relationship. This process is called as *Early Mesial Shift* (Fig. 7.19). In a closed dentition, this is not possible.

Replacement of incisors/incisal liability: The position of the dental lamina of the permanent teeth is located lingual to all the primary teeth. As a result, the incisors develop in their crypt lingual to and near the apex of the primary incisors. The permanent tooth resorbs the primary root

and erupts slightly labial to the location of the primary tooth (Fig. 7.20).

The mesiodistal width of the permanent incisors is larger than that of the primary incisors. Thus, the erupting permanent incisors require more space for proper alignment. This difference between the amount of space needed for the incisors and the amount available for them is called the "*Incisor Liability*". Incisor liability was described by Warren Mayne in 1969. A favorable incisal liability exists when the primary dentition is an open dentition. An unfavorable situation exists in closed dentition. The incisal liability is about 7.6 mm in maxillary arch and 6 mm in mandibular arch.

The space discrepancy is compensated by three mechanisms:

1. **Increased intercanine width:** During the period of permanent incisor eruption, significant amount of increase in intercanine arch width occurs. It is about 3 to 4 mm.
2. **Interdental spacing:** Spacing present in primary dentition helps in alignment of incisor. The primate space present in the upper arch mesial to primary canine is also used.
3. **Labial eruption of incisor:** Primary incisors stand upright. The permanent incisors, which replace them, are labially proclined placing them in a wider arch (Fig. 7.21).

Ugly Duckling Stage (Fig. 7.22)

This is a transient form of malocclusion wherein midline diastema is present between the maxillary central incisors. It is also called physiologic median diastema or

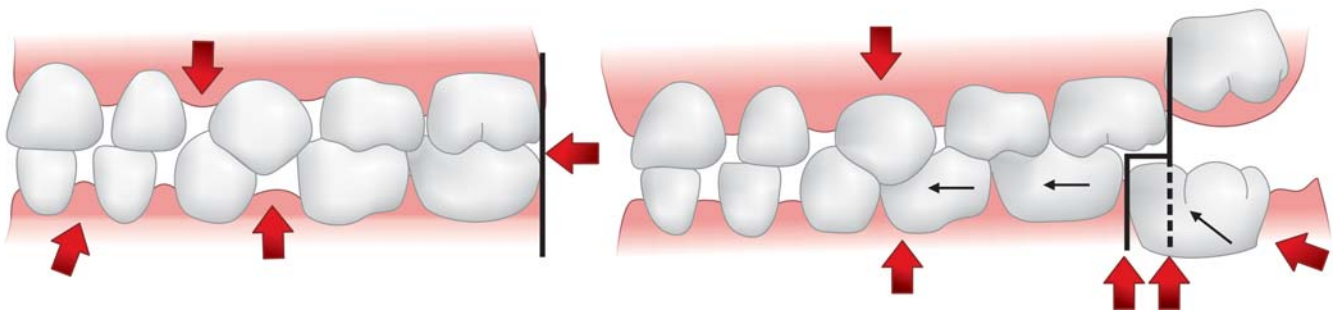


Fig. 7.19: Early mesial shift. The erupting mandibular first molar pushes the deciduous first and second molars which causes closure of primate space in the mandible

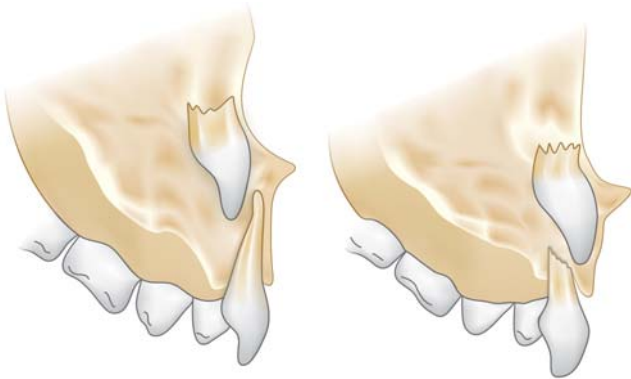


Fig. 7.20: Changing incisor position and labial eruption

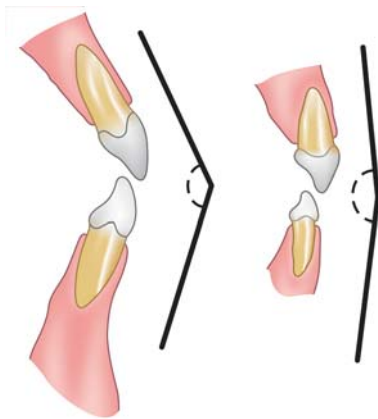


Fig. 7.21: Comparison of the labial inclination of permanent and primary incisors

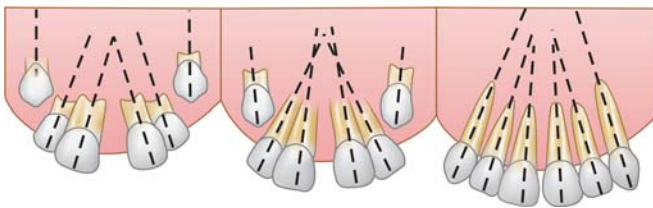


Fig. 7.22: Ugly duckling stage described by Broadbent at different ages. The median diastema closes spontaneously

Broadbent's phenomena. The ugly duckling stage is seen between 7 and 11 years of age. The mechanism of this stage is as follows:

During the eruption stages of the permanent canine, it impinges on the roots of lateral incisors. This pressure

causes the lateral incisor to erupt into the oral cavity with divergence of crown distally. Even after the lateral incisor erupts, this pressure effect from the erupting canine persists. This pressure is transmitted to the central incisors also, which causes the crowns to diverge and roots to converge towards the midline. This bilateral effect causes a midline diastema, which is transient. This temporary spacing that occurs between the central incisors and sometimes between central and lateral incisor gets closed automatically as the canine comes into occlusion. This stage is called ugly duckling stage because it represents a metamorphosis from an unaesthetic phase to an esthetic phase.

At the end of the first transitional stage, the molars usually erupt in end on relationship and the incisors are also present with slight crowding in the mandible and with spacing in the maxilla.

Second Inter-transitional Period

This period lasts from the complete eruption of incisors until the beginning of replacement of deciduous canines and first and second molars for approximately 1½ years. The vertical dimension of face increases which allows for heightening of the alveolar ridge. Space for maxillary and mandibular second molars is gained by bone remodeling in maxillary tuberosity and mandibular ramus. Maxillary canines are still developing and the premolars are placed between the roots of their respective predecessors.

Second Transitional Period

This active stage involves replacement of primary canines and molars. This exchange normally takes place between 10 and 12 years of age. The desirable sequence of eruption of permanent teeth is shown in Figure 7.23.

Leeway space of nance: The combined mesiodistal width of the deciduous canine, I and II primary molars is greater than the combined mesiodistal width of the permanent canine, I premolars and II premolars. This is called leeway space of Nance (Fig. 7.24). In the maxillary arch, it is about 0.9 mm on one side, totaling to 1.8 mm. In the mandibular arch, it is about 1.7 mm on one side, totaling to 3.4 mm. When the primary second molars are lost, there is an adjustment in the occlusion of the first molar teeth. There is decrease in arch length in both maxillary and mandibular arches as the first molars shift mesially.

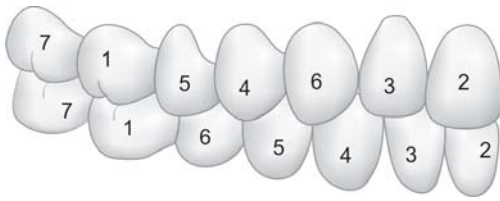


Fig. 7.23: Desirable sequence of eruption of permanent teeth

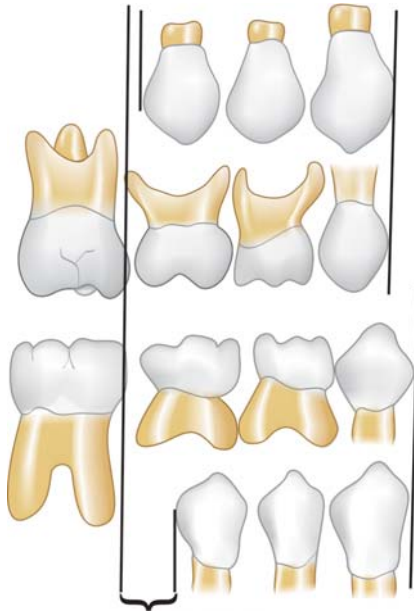


Fig. 7.24: Leeway space of Nance, it is more evident in mandible

This shift of molars is more in the mandible which accounts for the establishment of full cusp Class I molar relation from flush terminal plane relationship in deciduous dentition. This shift is called *late mesial shift of molars* (Fig. 7.25).

Change in molar relationship from mixed dentition to permanent dentition: There are two important contributors to the molar transition: (1) Late mesial shift of molar—after the shedding of primary II molar, the first permanent molar shift mesially. This mesial shift of the lower molar is more when compared to upper molar because of the more amount of leeway space. (2) Differential growth of mandible relative to maxilla is the second contributor. Because of the cephalocaudal growth, mandible grows more than maxilla.

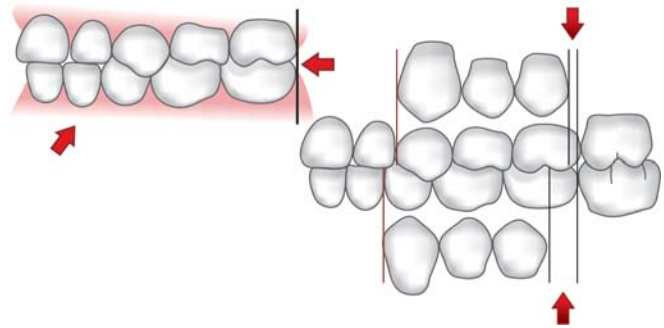


Fig. 7.25: Late mesial shift using the leeway space

Features of normal occlusion in permanent dentition includes:

Overlap: In normally occluding dentition, the maxillary teeth are labial or buccal to mandibular teeth.

Angulations: Permanent teeth have buccolingual and mesiodistal angulations.

Occlusion: With the exception of mandibular central incisors and maxillary third molars, each permanent tooth occludes with two teeth.

Arch curvature:

- Anteroposterior curvature in the mandibular arch — Curve of spee
- Corresponding curve in the maxillary arch is called compensating curve
- Buccolingual curvature from one side to other side is called Monson's curve.

Overbite: The normal overbite expressed in terms of percentage in adult dentition is 10 to 30 percent.

Overjet: Normal overjet is 1 to 3 mm.

Molar Relationship: Class I Molar — Mesio Buccal cusp of maxillary first permanent molar occludes in the Mesio-buccal groove of the permanent mandibular first molar.

The upper and lower molars occlude eventually in a manner determined by the spacing in primary dentition, terminal plane relationships, leeway space, differential growth of the lower jaw and muscular forces.

Dimensional changes in the dental arch during growth: In order to study the dimensional changes in dental arch, during growth, the following terms have to be understood (Fig. 7.26).

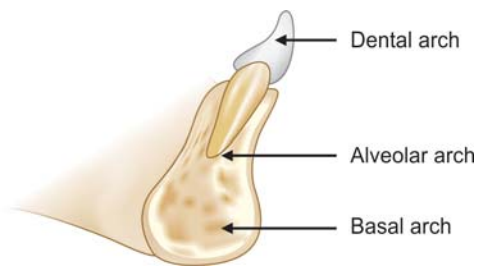


Fig. 7.26: Relationship of the three arches

Basal arch: This corresponds to the basal bone of maxilla and mandible.

Alveolar arch: Joins the tooth to the basal arch.

Dental arch: This denotes the combined mesiodistal widths of the teeth.

The basal arch is determined by the configuration of maxilla or mandible. Alveolar arch joins the tooth to the basal arch. For proper relationship there should be harmony among all the three arches. During growth, the values of alveolar arch and basal arch change, but the sizes of the teeth in the mesiodistal dimension remain the same. Routinely measured arch dimensions are (i) Width measured at the canines, premolars (deciduous molars) and first permanent molars; (ii) Length or depth and (iii) Circumference (Fig. 7.27).

Width: Dimensional increase in width involves mainly alveolar process growth and there is little skeletal width change.

There is greater correlation with maxillary arch width increase and vertical alveolar process growth. The dental arch width increase takes place due to vertical alveolar process growth because the maxillary alveolar process diverges during vertical growth. In the case of mandible, it is almost parallel (Moyers, 1976). The clinical significance of this is that the width of the maxillary arch can be easily altered for the purpose of treatment when compared to mandibular arch width.

Development and eruption of teeth also contributes to the increase in arch width.

In mandible, the increase in intercanine width is only marginal and it is the result of distal tipping of primary canine into the primate space as the incisors erupt.

Intercanine width is completed at 9 years of age in girls and at 10 years of age in boys. In maxilla, intercanine width is complete by 12 years in girls and at 18

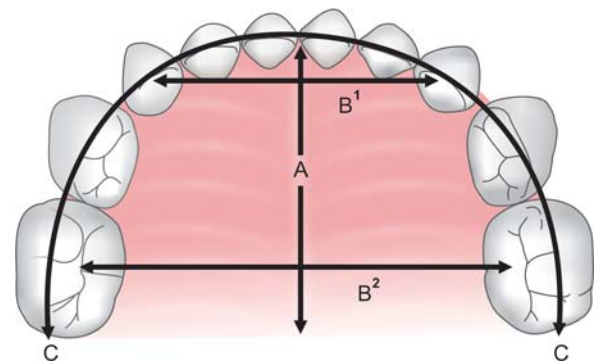


Fig. 7.27: Various arch dimensions. A arch length. B¹ intercanine width. B² intermolar width. C–C arch perimeter or circumference (Source: Moyers Handbook of Orthodontics)

years of age in boys. The delay in growth of maxillary intercanine arch width serves as a *safety valve* for pubertal growth spurts in mandible.

There is significant sex difference in the maxillary intercanine width increase.

The increase in width in premolar area is due to general widening of the arch due to increase in vertical growth. In the mandible, the increase is due to buccal placement of crowns of premolars. Increase in the maxillary premolar area width is more than in the mandible and it is also more in males as compared to females.

The mandibular molar width also is significantly less when compared to maxillary molar width. The decrease in molar width in mandible could also be due to mesial shift of the mandibular molars.

The important mechanism for postnatal increase in width is due to surface deposition on the lateral border of mandibular body or corpus.

The midpalatal suture can be reopened with rapid palatal expansion appliances and clinically results in larger amount of widening of maxilla.

Length or depth: Dental arch length or depth is the measured distance at the midline from a point midway between the central incisors to a tangent touching the distal surfaces of the second primary molars or second premolars (Fig. 7.27). The increase in anteroposterior length of the whole maxillary and mandibular arch length occurs due to resorption and deposition.

Arch circumference or perimeter: Arch circumference is the measured distance from the distal surface of the

second primary molar or mesial surface of first permanent molar around the arch over the contact points and incisal edges in a smoothened curve to the distal surface of the second primary molar or mesial surface of first permanent molar of the opposite side.

There is reduction in arch circumference with aging and it is more pronounced in mandible due to the following reasons:

- Increased mesial shift of the first permanent molars to compensate for the leeway space;
- Mesial drifting tendency of the posterior teeth throughout life;
- Interproximal wear of the teeth;
- Lingual positioning of the mandibular incisors due to differential mandibular growth;
- Tipped position of molars and incisors. Mesially tipped molars and distally tipped incisors contribute to the reduction in arch circumference;
- The role of third molars is debated and not conclusive.

The mandibular arch perimeter shows greater variability when compared to maxilla. The maxillary arch perimeter, on the contrary increases (Fig. 7.21) due to the labial angulation of the maxillary permanent incisors when compared to primary incisors.

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Chapter

8

Growth of Soft Tissues

CHAPTER OUTLINE

- **Significance of Studying Soft Tissue Growth**
- **Methods of Studying Soft Tissue Growth**
- **Soft Tissue Changes due to Growth**
 - Nose
 - Lips
 - Chin
 - Profile
 - Height
 - Depth
 - Oropharynx
 - Tongue
- **Soft Tissue Changes due to Treatment**
 - Extraction vs nonextraction
 - Retraction of maxillary incisors
 - Maxillary protraction therapy
 - Mandibular advancement and genioplasty
 - Double jaw surgery (maxillary impaction and mandibular advancement)
 - Bionator treatment

The form of human skeleton is beautified by the drape of soft tissue. Soft tissue comprises of not just the skin and subcutaneous tissue but also the underlying muscles. Muscles of the face are responsible for expressions like laughter, joy, sorrow, surprise, etc.; emotions that enliven human existence. Soft tissue drape adds esthetic dimension to the otherwise bare skeleton. Esthetic improvement is the single most important demand of the patients undergoing orthodontic treatment.

The thickness of soft tissue is not uniform throughout body, it varies markedly; soft tissue covering some of the cartilaginous structures like nose and ear is highly adherent but in other areas they are thick, e.g. lips. Another important aspect of soft tissue is that its growth does not follow the growth of the underlying hard tissue;

the soft tissue growth is highly independent. Infact, hard tissue growth depends on the soft tissue growth. Before 1950s, cephalometrics and its analyses giving importance to skeletal growth was used extensively. The study of soft tissue has gained momentum only after the 1950s. Angle had given significance to the profile of the patient before treatment, though not considering the soft tissue as a whole. His philosophy of nonextraction treatment had as one of its aims “arriving at a profile as straight as Apollo Belvedere’s” (Fig. 8.1). Angle considered the profile of Apollo to be ideal. Extraction treatment soon followed but to the dismay of many orthodontists, dishing in of the profile and worse esthetic outcome was noticed.

SIGNIFICANCE OF STUDYING SOFT TISSUE GROWTH

Pioneers in the study of soft tissue growth and behavior were Subtelny, Burstone, and Bjork. The turn of 20th century has seen the arousal of soft tissue paradigm. Increased emphasis on the role of soft tissue in deciding the treatment plan has been placed by Sarvar. Diagnosis and treatment planning are the keystones to orthodontic treatment and not the treatment procedure by itself. Hence, it is important to understand the role and importance of profile and soft tissue in arriving at a treatment plan. The importance of the position of nose and chin in relation to the lips is also realized. Burstone studied the lip posture in various malocclusions and in rest and occluded position. With years of study, it has become apparent that nose and chin continue to grow beyond the age of adolescence. Growth of nose is noticed at late adolescence in males. There is gradual retrusion of lips with continued growth of nose. The



Fig. 8.1: Apollo Belvedere

profile tends to flatten with age. According to Proffit, the orthodontist at the end of treatment should be able to place the upper lip vermillion beyond the soft tissue point A and lower lip should be as prominent as the chin. Nose-lips-chin relationship is an essential esthetic criterion. The profile of middle and lower third of face should always be considered before deciding on a treatment plan. Injudicious extraction of teeth in an already straight profile with a prominent nose will flatten/dish in the profile further. Nasolabial angle is another important esthetic criterion. An already obtuse nasolabial angle should make the examiner think twice before taking extraction decision. The recent trend towards esthetics among general population is the demand for fuller profile with thick lips. So already straight or slightly concave profile are best treated non-extraction attained by proclining the incisors to an esthetic limit. Esthetics is at the top of the priority list for most patients. In certain facial forms, e.g. patients with class II div I malocclusion, nose is very prominent and with growth the disproportionality in growth of nose is maintained. Extraction treatment planning will not only worsen the profile but also make the nose look even more prominent.

Growth of every individual is different but on a broad scale there is a general pattern of growth that we all follow. Growth of soft tissue similarly has a general trend and it is mandatory to study the growth of soft tissue to understand its behavior during treatment and to forecast the changes, to prevent any untoward treatment result due to faulty treatment plan. Some of the objectives

of soft tissue evaluation are as follows (Martha Meija et al, 2000).

- Retract, maintain, or protract upper and/or lower lip.
- Increase, maintain, or decrease vermillion display (lip thickness).
- Reduce lip strain, mentalis muscle strain, and interlabial gap or maintain lip competence.
- Increase, maintain, or decrease nasolabial angle.
- Increase, maintain, or decrease mentolabial angle.
- Increase or maintain cervicomental angle.
- Reduce, maintain, or increase the gingival display on smiling.
- Improve facial asymmetry.
- Increase, maintain, or decrease width of the alar base.
- Increase, maintain, or decrease the vertical and/or anteroposterior projection of the soft tissue chin.

METHODS OF STUDYING SOFT TISSUE GROWTH AND ASSESSMENT OF BALANCED PROFILE

Longitudinally soft tissue growth can be studied by the following methods:

- Cephalometrics
- Facial photographs both frontal and lateral
- Linear photogrammetric analysis
- Laser surface scanning.

Various profile lines are used to evaluate the face (Table 8.1). Ricketts defined the Esthetic plane (E-plane) as a line tangent to the chin and the tip of the nose. Gonzales-Ulloa suggested dropping a vertical line through soft tissue glabella to evaluate the position of the chin. Soft tissue pogonion should lie close to this line. According to Steiner, both the lips in well-balanced faces, should touch a line extending from the soft tissue contour of the chin to the middle of an 'S' formed by the lower border of the nose. This line is referred to as S-line. Merrifield's Z line is drawn tangent to the chin and the more protruding lip (usually the upper). The lower lip should lie on the line or slightly behind it. Holdaway's profile line extends from the chin through the upper lip and intersects the nose approximately 10 mm behind its tip. If the ANB angle is 2° , the profile line intersects the NB line at an angle of 8° . Bishara et al, found that Holdaway's soft tissue angle is an age-dependent measurement and progressively decreases from 5 to 45 years of age. Burstone measured facial convexity as the angle between glabella to subnasale and subnasale to

Table 8.1: Various profile lines used to evaluate balanced profile face

<i>Profile Line</i>	<i>Description</i>
Ricketts	Line tangent to the chin and the tip of the nose.
Gonzales-Ulloa	Vertical line through soft tissue glabella to evaluate the position of the chin. Soft tissue pogonion should lie close to this line.
Steiner	The lips in well balanced faces, should touch a line extending from the soft tissue contour of the chin to the middle of an S formed by the lower border of the nose. This line is referred to as S-line
Merrifield's Z line	Line is drawn tangent to the chin and the more protruding lip (usually the upper). The lower lip should lie on the line or slightly behind it.
Holdaway's profile line	Extends from the chin through the upper lip and intersects the nose approximately 10 mm behind its tip. If the ANB angle is 2°, the profile line intersects the NB line at an angle of 8°. Bishara et al, found that Holdaway's soft tissue angle is an age-dependent measurement and progressively decreases from 5 to 45 years of age.
Burstone	Measured facial convexity as the angle between glabella to subnasale and subnasale to soft tissue pogonion.

soft tissue pogonion. Burstone also defined two important angles, which are the nasolabial angle and the chin-throat angle, which are on average 114° in males, 118° in females, and 114° in males, 106° in females, respectively.

SOFT TISSUE CHANGES DUE TO GROWTH

Nose

Nose is the most prominent structure in the profile of the face and it continues to grow downward and forward at a proportionately greater degree than any other structure in the facial profile. Nose undergoes constant change and an increase in size of the nose is noticed in males till adulthood. Increase in nose size takes place both in the horizontal and vertical direction but it is the anteroposterior dimension of nose that provides a gender difference. Growth of nose occurs for a longer period in male than female. There is a spurt in growth of nose at puberty for boys. Girls do not seem to show this spurt at 12 years. A study by Prahl-Andersen et al, reveals a decrease in nasolabial angle with age which is more in girls than in boys. It is initially larger in girls than in boys.

Increase in length of the nose was the highest and most rapid. In the study for 9 years, it increased by almost 31 percent, similarly depth of nose also increased by 30 percent. The increase in overall facial length receives a major contribution from the increase of midface-length of the nose. Increase in length of the nose is about 1 to 1.5 mm per year, increase in width of the nose is about 0.5 mm per year. An analysis of growth changes

in soft tissue profile by Nanda et al, from 7 to 17 years divided the nose height into upper and lower nose height. The increase in the length of upper : lower nose height was in the ratio of 3:1. The increase in upper nose height occurred between 7 to 8 years, there was a decline in growth from 8 to 11 years and acceleration at puberty. Increase in upper and lower nose height is seen in both males and females but a significant increase in upper nose height occurred in males more than the females. Nose height attained adult value by around 15 years for females but only at 18 years for males. In a study by Subtelny, it was found that an average increase of 3 to 5 mm in the length of the nose is evidenced every three years.

Depth variation in nose is the main reason for profile changes in late adolescence and adulthood. According to Subtelny, increase in the nose tip is about 1 mm per year from 9 to 15 years of age. Anterior advancement in the position of nose tip is about 0.6 mm per year calculated from base of the nose. In a study of the development of the nose and the facial profile by Genecov in patients from age 7 to 18 years, it was found that nose grows greater in anteroposterior length, measured as projection from the soft tissue nasion, 5 to 6 mm in females from 7 to 12 years, but in males increase from 7 to 12 years is only about 4 mm. From age 12 to 17 years, males showed an increase in anteroposterior growth of about 4 to 5 mm while in females the increase was only 1 to 2 mm. Males continued to show growth in length of the nose even after 17 years of age. Ultimately, the nasal projection

in men is greater than women because men continue to show growth in the part even after the females have stopped growing. Nose depth (Nanda et al) attains 85 percent of adult value for males at 7 years but almost 90 percent of adult value for female children of the same age. The sagittal growth of the soft tissue of the nose is independent of the underlying skeleton and continues to grow even after the growth of the skeleton is complete. The ratio that for soft tissue to skeletal depth of the nose is 1:2 at 7 years becomes 1:1.5 at 18 years. According to Posen, changes in the form and size of the nose are found after the age of 13 years. There was no variation with gender in the rate of growth of nose in his study. Girls seem to have a more matured form of the nose than boys at all ages.

Buschang et al, studied the growth of the nose relative to skeletal maxillary growth in girls from 6 to 14 years of age. It was found that annual horizontal growth of nose tip—pronasale was 1.54 mm in childhood and 1.89 mm in adolescence, that for subnasale was 1.25 mm for childhood and 1.23 mm at adolescence. Growth of nose in relation to point A and ANS is not equal but closely related. Every mm of growth at ANS is associated with 0.6 mm per year of growth at Sn and Pr but growth at these soft tissue points is more relative to point A. Horizontal growth increments of nose at Pr in adolescence was found to be more than that in childhood.

Inclination of the nose between dorsum and PMV plane depends on the sagittal growth of the nose. Increase in the angle was more in males than females and the average adult value for males was larger compared to that for females. Bishara et al, when studying the facial profile changes from childhood to adolescence found out that total facial convexity increases with age (convexity of the profile from soft tissue point glabella, tip of nose—pronasale and soft tissue pogonion). In other words, angle decreases with age, becoming more convex. This is attributed to increase in the prominence of nose with growth when compared to other structures in the profile. The increase is seen both for males and females. A study by Chaconos shows that class II subjects were found to have more elevated nasal bridges. The shape of the dorsum also followed the profile; class II subjects had a more convex dorsum, class I subjects a straight one and class III subjects a concave dorsum (Fig. 8.2).

Lips

The contribution of lips to the profile is very important. Lip position can be manipulated with orthodontic treatment. Severe retrusion of lips due to excessive retraction of the incisors can worsen the profile. In studying the growth of lips, the following points should be noted: length of the lips, thickness of the lips, lips in profile, position or relation of the lips to reference planes and influence of various treatment procedures on the lips and interlabial gap before and after treatment.

Growth of the lips is found to follow the general body growth of Scammon's curve with the soft tissues and muscular tissues. According to Subtelny, the lips tend to grow at a gradual pace till 15 years of age following, which the growth rate slows. Lips also increase in thickness at the vermillion till 15 years: Thickness increase at vermillion is greater than that near points A and B for upper and lower lips respectively. The upper lips grow away from the palate and the lower lips grow away from the chin. Till what length the lips grow is a big question: Is there a genetically predetermined length which the lips try to attain? This query is answered by Subtelny by substantiating that lips always try to maintain a constant relationship with the position of the alveolar processes of the incisal region. There seems to be no vertical growth of the anterior alveolar process after the eruption of the central incisors and the distance between the alveolar margins as well as the vermillion border are maintained. Until adulthood, the upper lips appear to cover about 60 to 65 percent of the length of the upper incisors with the lower lips covering the rest. This constant relationship is slightly reversed at old age with the upper lip drape covering a longer portion of the upper incisor. The position of the lips anteroposteriorly depends on the underlying skeletal structures unlike the other soft tissue structures of the face that are largely independent of their hard tissue. When the alveolar processes and the teeth become protrusive so do the lips and vice versa.

When considering the length of the lips, upper lip length is measured from the subnasale to labrale superioris and subnasale to lower border of the upper lip. Similarly, lower lip length is determined from labrale inferioris to soft tissue gnathion or menton. According to rule of the thirds, the face is divided into upper, middle and lower thirds by four lines; a line across hairline, line through the superciliary arches, a line through the lower

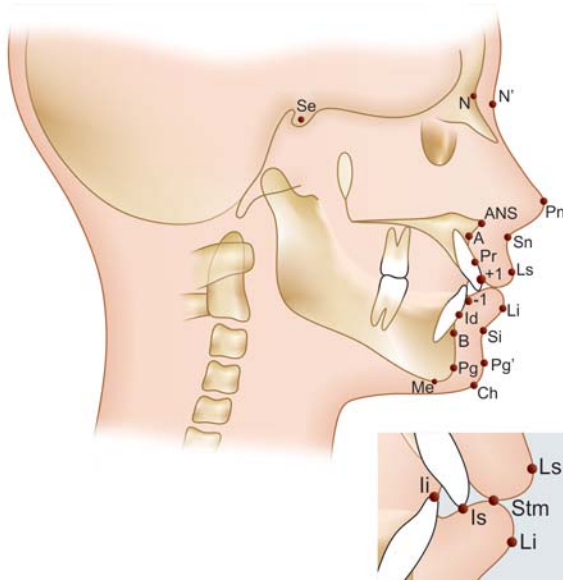


Fig. 8.2: Soft tissue landmarks (For description of the landmarks refer Chapter 11 on Cephalometrics)

border of the base of nose and a line through the lower border of chin. The lower third is again divided into an upper 1/3rd and lower 2/3rd. Normally upper 1/3rd is occupied by the upper lip and lower 2/3rd is occupied by the lower lip. This is an arbitrary method of assessing the lip length.

Length of the upper and lower lip increases from age 6 to 12 years in both boys and girls. An average value of 22.5 mm for boys and 20 mm for girls has been reported at age 12. Upper lip grows slightly in length with age, more in boys than in girls. The increase in length of lips has been attributed to an increase in facial height associated with growth. Increase in the length of the upper lip with treatment has a component of increase in bite height (opening of bite) associated with it. Burststone found out that annual increase in the length of the upper lip is about 0.2 to 0.3 mm per year; length increase in lower lip is about 1 mm per year initially which later tapers gradually. In a study of soft tissue growth in the 7 to 18 years age group by Nanda (AO 1990), the length of upper lip measured from subnasale to stomion was found to increase at an annual average value of 2.7 mm in males and 1.15 mm in females. Lower lip height (measured from lower lip stomion to soft tissue B point) increased by 4.2 mm in males and 1.5 mm in females. Increase in length of upper and lower lip in males is about twice the amount that occurs in females. Significant increase in length of the lips in males

may point towards a nonextraction treatment plan, whereas in females, there may not be an accountable change in protrusion of dentition and excessive gingival display.

Burststone reported that the increase in length of upper lip in class II cases was 1.9 mm and in class III cases, 0.9 mm. Increase in length of upper lip is more in class II malocclusion than in class III cases and the reverse is true for the lower lip. Increase in length of lower lip with age is more in class III malocclusion than that in class II. Though the increase in upper lip length is more in class II cases, class II div I cases are generally considered to have a short upper lip. In certain instances, this does not hold true. Burststone (AJO 1967) in analyzing the posture and length of the lips found that the distance between subnasale to vermilion of the upper lip does not show any appreciable change in different malocclusion groups. It may be short in certain subjects with class II div I malocclusion but need not necessarily be. An alternative method of assessing the lip length is to measure the relative lip length from the inferior border of the upper lip (stomion) to the incisal edge of the upper incisor (incision), in other words incisor display. The incisor display was found to be more in class II div I cases than in class I cases. This has important implications in treatment planning.

From childhood to adolescence, increase in the thickness of upper and lower lip is obvious; the thickness of upper lip is measured from the labial surface of the upper incisors to the most prominent part of the upper lip. The average thickness of upper and lower lips is 11.5 and 12.5 mm respectively. Increase in prominence of the incisors is associated with thin lips. Retraction of incisors by 3 mm increases the thickness of the lips by 1 mm. Thickening of lips with retraction is principally due to elimination of lip tension. Nanda et al, measured upper lip thickness at two points; at point A and labrale superioris. The increase in the thickness of upper lip at point A was 4.7 mm for males and 3.5 mm for females. The incremental thickness at labrale superioris was found to be 3.2 mm for males and 0.7 mm for female from 7 to 18 years. The increase in lip thickness was larger in males than females. Lower lip thickness was measured at labrale inferioris, and soft tissue point B. At labrale inferioris the lip thickness increased by 2.4 mm and 1.4 mm in males and females respectively. The horizontal measurement shows a spurt in boys after 12 years but girls do not show the spurt, the velocity of thickness

decreases after the age of 13 years. The peak value in lip thickness for females was attained by 13 years and for males by 18 years. The thickness at point B increased by 2.8 and 1.6 mm in males and females respectively. The lips became thicker in males after the age of 14 years.

There is relative retrusion of upper and lower lips with respect to Ricketts esthetic plane; with the lower lip closer to the E-plane than the upper lip. This is called *law of lip relationship*. This average decrease in the protrusion of lips is due to the fact that increase in depth of nose and chin is more than the increase in lip thickness. Upper lip recedes to about -4.2 mm in males (relative to E-plane) and -5.4 mm in females at 18 years of age. The lower lip position is -2.7 mm in males and -2.5 mm in females. Bishara measured the upper lip to be about 4 mm retrusive in relation to the E-plane for females and lower lip 2 mm posterior to the E-plane. Males have more protrusive upper lip than females. In the study from 5 to 17 years, it was found that lower lip became more retrusive with age in both males and females. The clinical significance is that patients in adolescence should never be treated to adult standards because more retrusion of lips can occur with age and growth.

Burstone in 1967, assessed the anteroposterior position of the upper and lower lips by relating them to the Sn-Pg' plane. A line drawn from the subnasale to soft tissue pogonion is used as the reference line. In a normal adolescent, the upper lip was found to be 3.5 mm beyond the Sn-Pg' plane whereas the lower lip was less protrusive, being 2.2 mm beyond the Sn-Pg' plane. In addition to nasolabial angle, inclination of upper lip was assessed by measuring the angle between the upper lip and the palatal plane. The normal angle was about 93°.

Chin

The prominence of soft tissue chin depends on the underlying skeletal chin. Soft tissue chin thickness is measured from pogonion to soft tissue pogonion (Pg-Pg'). Nanda et al, studying the soft tissue profile found out that thickness of chin in males is 2.4 mm while that in females is 1.5 mm. Prominence of chin is more in males than females. In other words, contribution of chin to a male profile is more than to a female profile. Chin is an important part of the profile that leads to straightening of the profile. Chin prominence increased at the rate of 0.2 to 0.7 mm per year. The value is slightly

greater for males. Subtelny measured the increase in soft tissue thickness over the chin to be 2.4 mm in males and 1 mm in females over a 15 year period. According to Singh (AJO 1990), the thickness of soft tissue chin varies with every facial type. Thickness of soft tissue chin was greater in brachyfacial type than the dolichofacials. The soft tissue over the chin is not even in thickness. The increase in soft tissue thickness after treatment is more in dolichofacial patients than brachyfacial patients. There was no change in chin thickness after 15 years in female subjects.

Profile

Profile changes during growth are different for hard and soft tissues. According to Subtelny, skeletal profile becomes less convex with age. With the continued growth of chin, the skeletal profile straightens but soft tissue profile remains slightly convex because of the continued growth of nose. This is an instance to prove that soft tissue growth is largely independent of skeletal growth. Convexity of the soft tissue profile is more prominent in females than males. The chin grows more in males than in females. Hence soft tissue profile of males is slightly straighter than that of females. According to Bishara, profile can be assessed by two parameters, (i) Total facial convexity and (ii) Facial convexity. Total facial convexity decreases with age as the nose prominence increases. Facial convexity assessed from glabella-subnasale-soft tissue pogonion increases from age 5 to 9 years, stabilizes from 9 to 12 years and decreases from 13 years to adulthood.

Nose continues to grow downward and forward. This is coupled with the forward growth of chin. These changes lead to retrusion of the lips relative to the disproportionate growth of the nose and chin. Though growth of all the tissues is more in males than females, the prominence of nose appears more in females than males because of the increase in the size of chin in males. This retrusion of lips relative to the other structures in the facial profile should be borne in mind when deciding the treatment plan. It is not uncommon to see patients who have prominent profiles in childhood develop better profiles in adulthood. In certain instances, nose is very prominent than the rest of the facial structures. Extraction of premolars and retraction of incisors will not only worsen the profile but make the nose to appear even more prominent.

Height, Width

Bishara analyzed the growth in height of soft tissues. Length of the face increases from childhood to adolescence. The study was conducted for 9 years and in that period, the facial length increased by 22.7 percent in females and 25 percent in males. The study was conducted on facial photographs of boys and girls at different ages (4 to 13 years). The assessment was made of the facial profile and frontal view photographs. Some of the parameters assessed were length of the face, nose, upper and lower lip; width of the face, nose, eyes, lips; and depth of the nose. Increase in length of the nose was largest and most rapid. Increase in width is more in females than in males, an average of 1 mm per year. Bizygomatic width increases about 9.5 percent in females and 8.8 percent in males. Intergonial width also increases but the rate of growth slows after the first year.

According to Ricketts, all the facial structures grow in proportion to each other and they follow the divine proportion. Width of the nose: width of mouth (between commissures): width between outer canthi of the eyes: width of face between the brows grow according to the divine ratio of 1:1.618. Vertically, the distance from alae to soft tissue chin and length between alae and trichion follows the golden rule. If the length of alae to upper lip is taken as 1, then the distance of lower lip to chin and from alae to eye is 1.6 (Fig. 8.3).

Depth

Increase in depth of the face measured from the outer canthus of the eye to porion was at the rate of 1 mm per year. Percentage increase is about 16.2 percent and 18.5 percent in girls and boys respectively. Anterior depth (glabella to outer canthus of eye) of the face increased at the rate of 1.3 mm per year. Increase in width of nose is 0.5 mm per year. Increase in depth is about 0.6 mm per year.

Oropharynx

Pharynx (naso and oropharynx) is associated with many functions, the most important of them being respiration. Growth of pharynx should be studied to understand the changes that take place in this functional space and find out the etiology for reduction in the volume of this space and its associated deformities. The most important and extensively researched functional abnormality

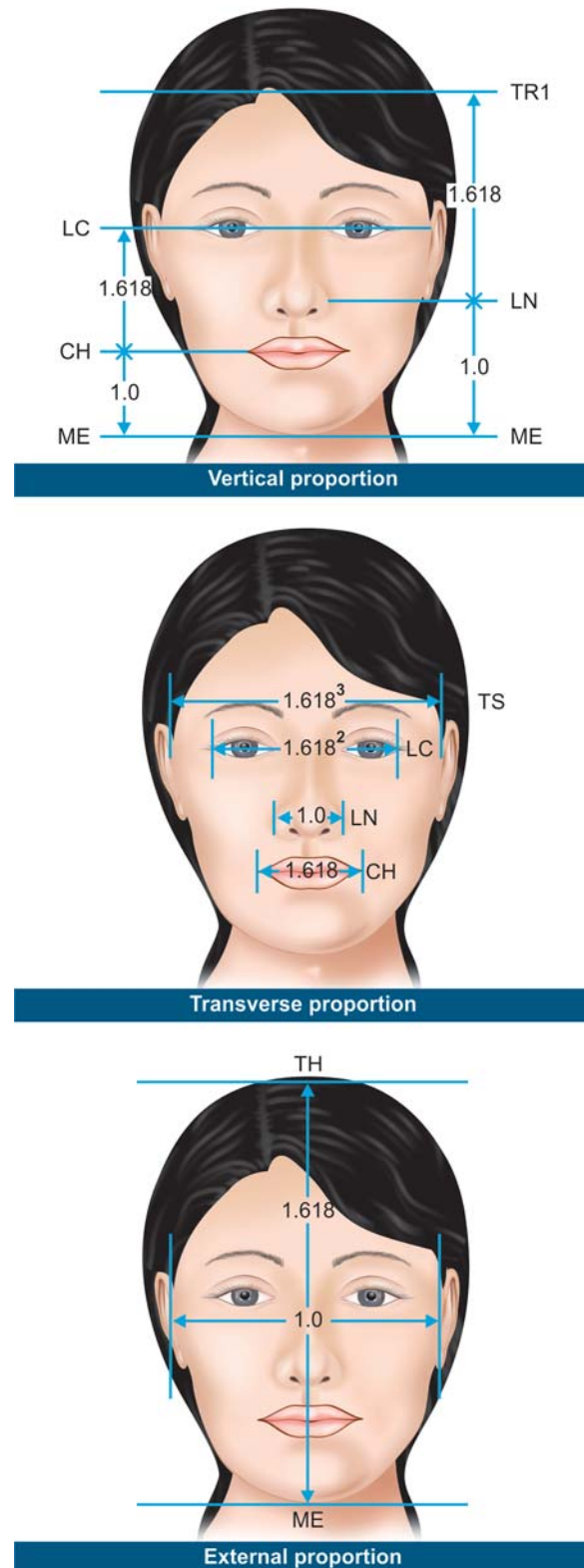


Fig. 8.3: The golden-ratio proportions of the ideal face

associated with pharynx is obstructive sleep apnoea. Etiologies to this problem are numerous but the basic cause is the reduction of pharyngeal space (retrognathic mandible, tongue obstruction, etc.). Taylor et al (AO 1996), studied the growth of oropharynx by means of a cephalometric analysis. Pharyngeal depth (antero-posterior) was measured at various levels. It was found that pharyngeal depth increased from the age 6 to 9 years, was stable from 9 to 12 years and again increased from 12 to 15 years; increase from the age 15 to 18 years is only meager. The increase from 6 to 9 years has been attributed to normal growth of pharynx, regression of adenoid tissue and in few cases surgical removal of adenoids has paved way for the increase in pharyngeal space. There is a plateau in the growth of pharynx from 9 to 12 years. There is another surge in the growth of pharynx from 12 to 15 years that is attributed mostly to the pubertal spurt in growth. It can also receive contribution from the complete regression of adenoids at this age.

It may be argued that pharyngeal constriction may occur after mandibular setback surgery but Saitoh (AJO 2004) proved through his study that pharyngeal adaptation occurred after few months of mandibular setback and there was no major airway restriction.

Tongue

Tongue is an important functional organ. It seems to follow the neural growth curve of Scammon's growth curve. Tongue at birth is so big that the infant is seen protruding the tongue out of its mouth almost all the time. The mandible at birth is small and it appears as if there is not enough oral volume to accommodate the tongue. With growth, tongue appears to recede inside the oral cavity; this is due to growth of the jaws that increase the intraoral volume. Tongue balances the muscle pressure from inside.

SOFT TISSUE CHANGES WITH TREATMENT PROCEDURES

Extraction vs Nonextraction

Bishara et al, conducted a study comparing post-treatment changes in the soft tissues of the face in extraction and nonextraction groups. It was revealed that upper and lower lips became more retrusive with extraction treatment especially extraction of first

premolars than nonextraction group. Upper lip length increased more among males and females treated by non-extraction. Similarly, vermilion height also increased in cases treated with nonextraction. Nasolabial angle became more obtuse among female patients treated by extraction. Bravo et al, on a study on the behavior of soft tissues after premolar extraction and orthodontic treatment concluded that retraction of upper and lower lips occurs by 3.4 and 3.8 mm to E line respectively. Nasolabial angle increases by 3.7°. Upper and lower lip protrusion relative to the Sn-Pg' line reduced by 2.4 and 3.1 mm respectively. Patients with lip protrusion of less than 2 mm relative to Burstone's plane and nasolabial angle greater than 110° should not be treated by extraction as far as possible.

Retraction of Maxillary Incisors

Talass studied the profile changes following incisor retraction. Nasolabial angle increased after retraction. Upper lip retraction with incisor retraction was found to be at the level of 1/5th of incisor retraction. Upper lip length did not change with treatment but length of the lower lip increased after treatment. There was increase in lower facial height. Reduction in interlabial gap was due to increase in the lower lip length.

Maxillary Protraction Therapy

Peter Ngan in his study on soft tissue changes after maxillary protraction found that profile straightened from being concave after protraction. Upper lip thickness reduced and lower lip thickness increased. Upper and lower facial heights increased. Inclination of lower lip decreased, and curvature of the lower lip increased. Inclination of lower lip became flatter but not to a significant extent as the upper lip.

Mandibular Advancement and Genioplasty

Ewing and Ross (1992) studied the changes in soft tissue profile with mandibular advancement and genioplasty; they also compared the hard and soft tissue movement with the surgeries. The result showed that both hard and soft tissue advanced in the ratio of 1:1 following mandibular advancement. The forward movement was very consistent. The hard and soft tissue ratio for genioplasty was also 1:1 but the result was not consistent, it reduced to 1:0.9. The lower lip advanced in the genioplasty group at the ratio of 0.5:1 of hard tissue

changes. The lower lip also thinned in the genioplasty group more than the nongeniotomy group. Veltcamp et al (AJO 2002) are also of the same opinion but they include a slight thinning of soft tissue chin thickness with advancement.

Double Jaw Surgery (Maxillary Impaction and Mandibular Advancement)

Jensen et al, analysed the soft tissue alterations after bimaxillary surgery and found that upper lip moved forward at a percentage of 90 percent of the total hard tissue movement. Upper lip shortened by 20 percent of the hard tissue impaction. Mandibular advancement produces 100 percent forward movement of soft tissue at pogonion but only 70 percent at the lower lip. The upper lip thinned by 1.5 mm at the labrale superioris.

Bionator Treatment

Lange et al, conducted a study to analyze the changes in soft tissue profile of patients treated with bionator starting in the mixed dentition for a period of 18.7 months on an average. There was forward movement of soft tissue pogonion in both bionator and control group but movement of Pg' is 1 mm more in bionator group than control group. There was also significant increase in anterior and posterior facial heights in bionator groups. Decrease in facial convexity G-Sn-Pg' was about 2.22°. Protrusion of the upper lip reduced by 1 mm in treated group. There was reduction in the protrusion of A' point and labrale superioris in the bionator group. Lower lip showed the most significant change, uncurling of lower lip occurred. There was forward movement of the lower lip with reduction in the labiomental angle. Lip length increased by 2.5 mm and lip thickness decreased by 2.6 mm.

Customized treatment planning is the order of the day and when the patients' demand is facial esthetics, a priority list with patients' demand for esthetics at the top of treatment plan is truly justified. When there is so much emphasis on final esthetic outcome of the treatment, it is mandatory to study the growth of the soft tissue, its change with age and its behavior to various treatment modalities. Most of the patients come to orthodontists at the start of pubertal spurt or late childhood. Before deciding on the treatment plan, the basis of soft tissue alteration and an idea of what the child will look like, decades after treatment, should be

borne in mind. Incisor display may reduce with age in boys due to increased lip lengthening than in girls. This gender difference should also be remembered. And there is also an amount of thinning of lips with age, which should be remembered before extracting teeth for orthodontic treatment. Soft tissue paradigm is the order of the day.

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Chapter

9

Adolescence and Craniofacial Growth

CHAPTER OUTLINE

- Endocrinology of Adolescence
- Timing of Puberty
- Pubertal Growth Spurt
- Clinical Features or Physical Changes of Pubertal Development
- Facial Growth During Puberty
 - Growth of cranial base during adolescence
 - Growth of maxilla during adolescence
 - Growth of mandible during adolescence
 - Soft tissue changes during adolescence

Adolescence or puberty is the period of life which leads to adulthood through dramatic physiological and psychological changes. Adolescence or puberty is also that period of development during which mammals typically acquire their reproductive capability. Somatic structural alterations of the body generally occur in synchrony with its physiological changes. Adolescence is characterized by increased growth rate in practically all the bones and muscles of the body. The amount of growth at adolescence is considerable. The composition of the body and face changes dramatically during adolescence. The parameter of growth rate and development of the facial skeleton is comparable with that of the somatic skeleton. Acceleration and depression in the growth of the jaws can be correlated with somatic skeletal growth rates. Hence, the clinical orthodontic considerations associated with pubertal growth spurt are concerned with the amount of growth and its effect on the outcome of treatment.

ENDOCRINOLOGY OF ADOLESCENCE

Puberty is a period during which many dramatic hormonal changes occur. Of these, it is clear that changes

in the axes controlling the secretion of growth hormone and gonadal steroids play central roles. Puberty is the maturation of the reproductive system and attainment of fertility. Initiation of puberty is controlled by the neuroendocrine system. It involves increased secretion of gonadotropin releasing hormone, gonadotropins, sex steroids, growth hormone and somatomedin C. The testosterone concentration in boys and, less dramatically, estradiol concentration in girls are higher in infancy than during childhood. Some sort of CNS restraint mechanism develops in late infancy which suppresses the reproductive system until puberty. The secretions are brought out by the changes in the hypothalamus-pituitary-gonadal axis. This axis functions at a lower level during infancy and childhood until puberty.

The onset of puberty is preceded by an increase in the androgen levels secreted by the adrenal glands. Adrenal androgens (androstenedione, dehydroepiandrosterone, and dehydroepiandrosterone-sulfate) are secreted in small amounts during infancy and early childhood, and their secretion gradually increases with age, paralleling the growth of the zona reticularis. The onset of DHEA-S production from the adrenal zona reticularis leads to the phenomenon of *adrenarche*. The latter occurs only in human beings and some old world primates, such as the chimpanzee, and in order to occur, a specific cell type with the capacity to synthesize DHEA-S must arise within the zona reticularis of the adrenals. The mechanism(s) by which this zone develops with age and the regulation of its secretion is not fully known. During this process, the plasma concentration of the adrenal androgens increases, whereas those of cortisol remains stable, suggesting that factors other than corticotropin are involved. These may include the elusive

androgen-stimulating factor, the existence of which has been repeatedly questioned. A role for corticotrophin releasing hormone (CRH) has also been proposed in the regulation of DHEA production, particularly in the human fetal adrenal gland. More recently, candidate hormones related to body mass, such as insulin and leptin, have been suggested as the triggers of adrenal growth and adrenarche. The onset of puberty is initiated by the maturation of the hypothalamic pituitary complex and input of the central nervous system which is called as the *gonadostat*. Prior to the maturation of the gonadostat the adrenal androgens appear to be transformed into estrogens in the peripheral fatty tissues, causing maturation of the gonadostat. The adrenal cortex begins secreting significant levels of the androgenic hormone dehydroepiandrosterone (DHEA) and its sulfated derivative (DHEA-S) approximately at the ages six to seven. Parker and Mehesh postulate that this early rise in adrenal androgens, termed adrenarche, stimulates hypothalamic maturation and initiates anabolic growth of the musculoskeletal system. Adrenarche occurs approximately two years prior to onset of puberty. Adrenarche is a morphologic change that occurs in the adrenal cortex, in which there is disappearance of the thin zone of connective tissue separating the adrenal cortex from the adrenal medulla and the zona reticularis becomes apparent. Androgenic hormones such as DHEA-S stimulate growth and proliferation of growth cartilage cells of the epiphysis and then potentiate the action of growth hormone. It was also found that growth hormone levels do not fluctuate during puberty and that children with adrenal insufficiency demonstrate delayed puberty.

This rise in the DHEA may be responsible for juvenile acceleration of growth which is more prominent in girls. The increase in androgen levels occurring in childhood is responsible for the appearance of body odour, and pubic and axillary hair. Although the temporal relation between adrenarche and the onset of puberty suggested that adrenal androgens might have a regulatory influence on the timing of puberty, it is now evident that the two events are independent processes.

Gonadotropin releasing hormone (GnRH), a decapeptide secreted by approximately 1000 neurons located in the basal forebrain and extending from the olfactory bulbs to the mediobasal hypothalamus, is responsible for the gonadotropin secretion by the

pituitary gland. Two types of neurons have been identified to date, GnRH neurons I, and II. The latter, have no known function in humans, and are not involved in reproductive function. GnRH neurons I originate in the embryonic period and exhibit an endogenous secretion very early in development. After birth, their activity is turned-off by the low circulating levels of androgens/estrogens released by the gonads, by means of a negative feed-back mechanism. At puberty, the reactivation of this *gonadostat* is independent of the effect exerted by the steroids, and is related to a reduced sensitivity to their action. GnRH stimulates the release of LH and FSH from the pituitary which in turn stimulates the gonads. LH and FSH have negative feedback effects on the hypothalamus, whereas testosterone (T) and androstenedione (A) produced by the testis, and estradiol (E2) produced by the ovary, inhibit both the hypothalamus and the pituitary gland. Inhibin, activin and follistatin also have feedback effects on both levels. GnRH secretion by the hypothalamus is under the control of a plethora of central and peripheral signals like excitatory aminoacids and other neurotransmitters such as GABA, gonadal sex steroids, adrenal and thyroid hormones, the GH-IGF-IGFBP axis, nutrition and related hormones such as leptin and insulin.

The onset of puberty is marked by the increase in secretion of the gonadotropin releasing hormone by the hypothalamus (Fig. 9.1). Increased secretion of the GnRh results in increased responsiveness of the pituitary

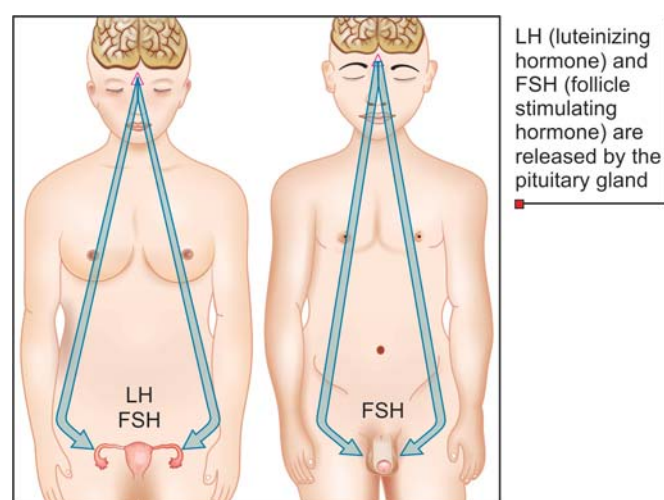


Fig. 9.1: Pituitary control of gonadotropins, namely luteinizing and follicle stimulating hormones

to GnRH, which leads to raised levels of gonadotropins, namely, follicle stimulating hormone (FSH) and the luteinizing hormone (LH) resulting in gonadal maturation and sex steroid secretion which result in accelerated growth, physical maturity and fertility.

The hypothalamus-pituitary-gonadal axis has negative control feedback mechanism. Increased levels of the sex steroids suppress the secretion of the gonadotropin releasing hormone from the hypothalamus. Normally, increased serum levels of the gonadal steroids, through the feedback mechanism, decreases the secretion of the gonadotropin releasing hormone. This feedback mechanism functions at a lower level. Gonadotropins and sex steroids are very low and there are minimal or no sex differences.

Regulation of the hypothalamic-pituitary-gonadotropin system is controlled by its maturational level and hormonal sensitivity before the onset of puberty (Fig. 9.2). In the pre-pubertal period, low levels of estrogen provide feedback to the hypothalamus which inhibits the secretion of the gonadotropin releasing hormone. With age, the hypothalamus becomes less sensitive to low levels of the sex steroids and therefore is able to secrete releasing hormones. The levels of estradiol and testosterone present in the pre-pubertal

individual are of adrenal origin and are not produced by the immature gonads. Though the levels are low initially, they rise steadily in order to stimulate the ovarian and testicular development, but measurable levels are seen only before the onset of puberty. All the mammals have the necessary cellular apparatus to secrete gonadal steroids prior to puberty; their levels are zero until after three years of puberty.

This regulation is the interplay between the levels of hypothalamic releasing hormones, pituitary gonadotropins and the gonadal sex hormones and alterations within the central nervous system. Before puberty, the hypothalamus is sensitive to the circulating levels of the sex steroids and thus the secretion of the releasing hormones is being suppressed. Therefore with maturity which is related to adrenarche (maturation of adrenal cortex), there is increase in the circulating levels of the gonadotropins. Recently three transcriptional factors, Oct-2, TTF-1, and EAP-1, have been identified as potential regulators of the cell network which controls the GnRH secretion. They regulate the expression of genes involved in cell function and cell-cell communication.

At puberty, the concentrations of two negative regulators of FSH secretion, inhibin and follistatin, changes in opposite directions, whereas the blood level

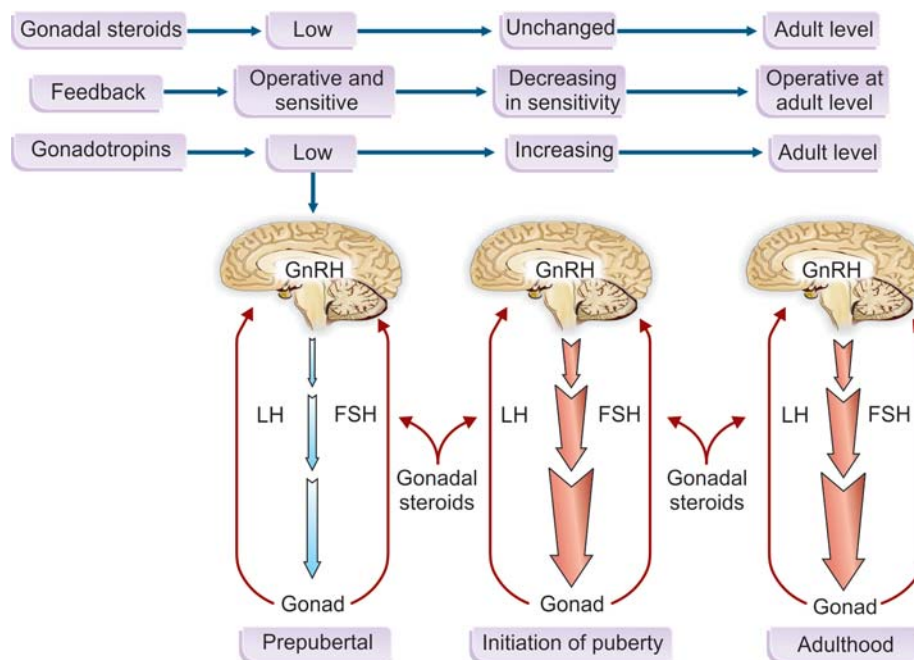


Fig. 9.2: Hormonal changes during puberty

of a positive regulator, activin A, increases, at least in females. All together, these alterations in serum concentrations of FSH-regulatory peptides lead to an increase in FSH secretion. Other hormones have been shown to undergo significant changes at puberty. Growth hormone (GH), insulin, insulin-like growth factor (IGF)-I, and its major binding protein, IGFBP-3, normally rise at puberty. The increase in growth hormone and IGF-I concentrations is probably responsible for most of the metabolic changes observed during puberty, including insulin-resistance, increased β -cell response to glucose, and the growth spurt.

TIMING OF PUBERTY

There is great variation in the timing of puberty in both males and females. The adolescent growth spurt generally occurs earlier in females than in males. The females precede males by 2 years in pubertal growth spurt.

During the pubertal growth spurt, the increased level of sex steroids increases the rate of skeletal maturation by increasing the rate of transformation of cartilage to bone. This acceleration in transformation of cartilage to bone is rapid and more prominent in girls. However, boys are slow growers but have a longer period of adolescence. They grow slowly and steadily during the pubertal period. The epiphyseal plates close more slowly in males than in females. The cutoff timing in puberty also seems to be well defined in females when compared to males.

Genetic and environmental factors also affect the timing of puberty. There are late and early maturing families present. Ethnic influences and the body type also affect the timing of puberty. Presence of certain amount of fat is required for the onset of puberty. For instance, girls with a slender body type mature late (e.g. athletic girls). Other factors include seasonal and cultural factors; during spring and summer, growth tends to be faster. Children in urban areas tend to mature earlier than those in rural areas.

PUBERTAL GROWTH SPURT

During the childhood the linear growth velocity decreases steadily throughout childhood. At approximately 10 years of age in girls and 11.5 years in boys, growth rate reaches the lowest level. This is called as the *minimum peak height velocity (MHV)* or *growth height velocity*. This

is the reference point for the beginning of the pubertal growth spurt. The maximum peak height velocity occurs at about 12 and 14 years in girls and boys, respectively. The sesamoid bone of the thumb appears approximately six months before the peak height velocity. In both sexes, the total duration of the pubertal spurt averages about 5.2 years. The maturation of the neuroendocrine system concerned with puberty takes place even before the physical signs of puberty are evident. In females, peak height velocity occurs even before the menarche. Peak height velocity occurs later in boys as there is continuous increase in luteinizing hormone and testosterone throughout.

CLINICAL FEATURES OR PHYSICAL CHANGES OF PUBERTAL DEVELOPMENT

Clinically, the onset of puberty is announced by an increase in the body stature and the appearance of secondary sex characteristics, in particular, the appearance of breast in females, testicular enlargement in males, and pubic/axillary hair in both sexes. These features evolve from their initial appearance till the attainment of adulthood, and their development is rated into 5 stages according to Tanner's criteria.

Tanner's Criteria for Females: Breast development in females may be unilateral for several months, and begins with an elevation of the breast and papilla, and a slight enlargement of the diameter of the papilla (stage 2) known as the breast bud. The breast and the areola enlarge further (stage 3) until the areola and the papilla form a secondary mound above the level of the breast (stage 4). The mature stage (stage 5) occurs at the end of puberty or with the first pregnancy and is characterized by the projection of the papilla only, due to a recession of the areola in relation to the contour of the breast (Fig. 9.3). Pubic hair in females appears sparse, long, slightly pigmented and curly, mainly along the labia (stage 2). It becomes progressively darker, coarser and curlier and spreads over the junction of the pubes (stage 3) progressively but covering a smaller area than in an adult (stage 4). In the adult stage, the hair is distributed as an inverse triangle and spreads to the medial surface of the thighs (stage 5) (Fig. 9.3). Pubarche is usually preceded by the appearance of the breast bud.

In males, the penis and pubic hair usually mature simultaneously as both processes depend on circulating

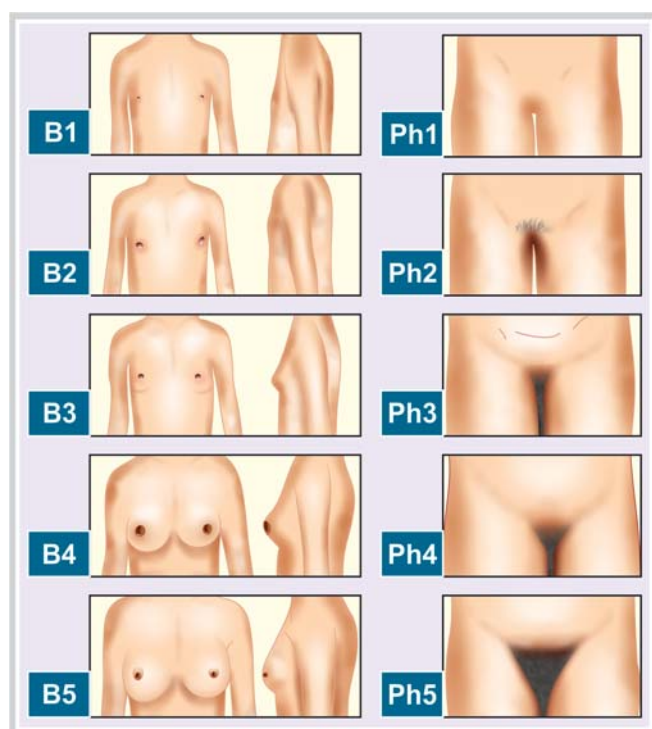


Fig. 9.3: Stages of breast development. B1: pre-pubertal; B2: breast bud; B3: enlargement of breast and areola with no separation of the contours; B4: projection of areola and papilla to form a secondary mound above the level of the breast; B5: recession of the areola to the general contour of the breast with projection of the papilla only. (right) Stages of pubic hair development in females. Ph1: pre-pubertal; Ph2: sparse growth of long, slightly pigmented hair, usually slightly curly, mainly along the labia; Ph3: the hair is darker, coarser and curlier and spreads over the junction of the pubes; Ph4: the hair spreads covering the pubes; Ph5: the hair extends to the medial surface of the thighs and is distributed as an inverse triangle.

androgens. Initial signs of puberty includes fat spurt in the boy as he gains weight with a feminine fat distribution, which is due to estrogen production by Leydig cells in the testes. These cells are activated before the Sertoli cells produce testosterone.

The stages of development are, however, rated independently to establish potential disorders of the testes and adrenal glands (Fig. 9.4). The onset of puberty in boys is marked by testicular enlargement. Usually, pubic hair in boys appears initially on the scrotum and at the base of the penis and develops to the adult stage progressively as in females, with a final distribution as an inverse triangle. Furthermore, during puberty, the

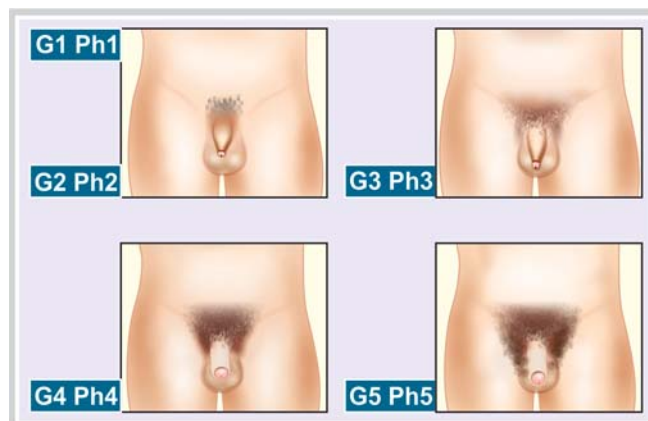


Fig. 9.4: Stages of pubic hair and genital development in the male. G1: pre-pubertal; G2: the testis and scrotum enlarge, and the skin of the scrotum shows some reddening and change in the texture. Sparse growth of pigmented hair, usually slightly curly, mainly at the base of the penis (Ph2); G3: Testis and scrotum enlarge further, the penis grows mainly in length but also in breadth. The hair is darker, coarser and curlier and spreads over the junction of the pubes (Ph3); G4: Scrotum, testis and penis grow further with development of the glans and further darkening of the scrotal skin. The hair spreads covering the pubes; G5: adult stage with spreading of the hair to the medial surface of the thighs.

membranous and cartilaginous components of the vocal cords lengthen; facial hair appears initially at the corners of the upper lip and the upper cheeks and spreads to the rest of the face and chin after Tanner stage 5.

FACIAL GROWTH DURING PUBERTY

Growth of the jaws usually correlates with growth in the body height and events in puberty. The growth of the mandible is not as dramatic as increase in the body height and there is only modest growth of maxilla. The cranial base also shows a spurt during the pubertal growth spurt. The cephalocaudal growth gradient is dramatically evident at puberty. In the face, more growth occurs in the lower jaw than in the upper jaw. Nanda, in his study (1955) observed that an increase in the circumpubertal growth velocity and growth of the face preceded the peak height velocity by nine months. He also stated that it was possible to predict the time of onset of adolescent growth spurt in the face from the acceleration of growth in body height. Hunter argued that the maximum height velocity coincided with the maximum facial growth.

Anibal M. Silveira, Leonard S. Fishman, J. Dani demonstrated that the mandibular growth rates of early and late maturers are significantly different during the late stages of pubertal growth. Late maturing individuals showed larger growth increments as compared to average and early maturing individuals. This study also supported the finding, that there is a difference between the growth of the mandible and the maxilla during the late stages of the pubertal growth spurt. The mandible grew significantly more than the maxilla.

Growth of Cranial Base During Puberty

The cranial base also shows some increases in growth increments during puberty. Hunter was the first one to study about the spurt in cranial base growth during adolescence. He stated that there is an acceleration of the growth of the Ba-S distance at the age of 12.5 years and an increase in the S-N length at the age of 8 to 15 years. Later, in a study by Arthur Lewis in 1974 on the growth increments of cranial base in puberty in boys, it was seen that acceleration in the growth of the lengths S-N, Ba-N, Ba-S was seen. The spurt in Ba-N length is smaller than S-N in early maturing boys. The Ba-S has greater spurt in shorter boys but the Ba-S length is greater in taller boys. The pubertal growth spurt in the cranial base usually precedes the peak height velocity. The spurt in the cranial base length is mainly due to the growth in the sphenoccipital synchondrosis. Contribution of the growth of the cranial base length at the foramen magnum and nasion is minimal and hence the growth is mainly due to the growth at the synchondrosis. Lewis studied the sex differences in the pubertal growth spurt in cranial base: it is much earlier in girls than in boys and it is greater in boys than in girls.

Growth of Maxilla During Puberty

There is only modest increase in the growth of maxilla during puberty. The postnatal growth of maxilla involves remodeling and displacement, and the cartilaginous growth of the nasal septum. Very few people have studied the growth of maxilla. Singh and Savara, and Bjork are the only people who made longitudinal cephalometric studies regarding the growth of maxilla. O' Rielly used the data from the studies of Bjork to calculate the increase in the length of maxilla during puberty. He concluded that the maximum increment in maxillary length occurred

before as well as after menarche, the onset of epiphyseal-diaphyseal fusion, and the peak of growth in height. The later the maximum occurred, the earlier the onset of menarche occurred in relation to the peak.

The amount of peak and duration of the growth were not dictated by the absolute size of the maxilla at its peak of growth. There was no significant difference in the amount of growth of maxillary length before or after menarche. Timing of maximum increment in maxillary length was weakly correlated with the onset of epiphyseal-diaphyseal fusion and menarche.

Pubertal Growth of Mandible

The mandible follows the cephalocaudal gradient of growth and follows the growth of the body height. In his longitudinal cephalometric study, Nanda (1955) stated that between the ages of 13 and 16 years of age there was a significant increase in the size of the mandible. Sella-gonion shows a greater proportionate increase than gonion-gnathion and, while gonion-gnathion stop growing by 19 years, sella-gonion continues to increase. In addition, it will be seen that, for the boy under discussion, all except two dimensions are growing even at the age of 20 years.

Bjork evaluated the growth of the condyles in 45 boys between 7 and 21 years of age. Bjork found that only 11 individuals (less than 25% of the sample) had what he was able to describe as a "discernible pubertal growth variation". For these 11 subjects, he described a slower growth rate around 12 years of age amounting to a mean of 1.5 mm, and a "spurt" two years later that averaged 5.5 mm and ranged between 4.0 and 8.0 mm. For the rest of the 34 subjects in the study, there was a steadier annual condylar growth averaging 3.0 mm during the same period. As for the timing of the spurt, the mean age for its occurrence was 14.0 years with a range between 12 and 15 years. Bjork's conclusions indicate that there was a discernible but not significant spurt in mandibular growth during puberty and there was no relationship between the intensity and direction of growth.

The Michigan growth studies and Bolton growth studies showed only gradual increase in size of the mandible with age. They did not demonstrate any significant pubertal spurt in the growth of the mandible. The explanation could be that the earlier studies had samples divided into those who actually demonstrated

pubertal growth spurt in mandibular growth and those who did not show any such change. Another reason would be that when the scale of the graph is enlarged, even a small change would appear big. Hence, only about 25 percent of the people exhibited acceleration in the growth of the mandible during puberty. From the above studies it was inferred that it is not wise for us to wait for the spurt in mandibular growth and then plan the treatment. This does not mean that no such spurt occurs. The spurt occurs but not in a consistent and even manner. The main problem for the clinician is to identify the person in whom the spurt occurs and also its duration and extent.

In the Iowa growth samples, the increase in mandibular length is seen in three groups, each of two years duration designated as, premaximum growth period, maximum growth period and post maximum period. The average changes in mandibular length were: 6.3 mm, 5.4 mm, and 3.7 mm, respectively. Thus, there is significant growth of the mandible which takes place over a longer period during adolescence. However, this does not assign a specific time for early and late treatment as the timing of treatment is also affected by numerous other factors.

Annual rates of vertical growth of mandible range between 0.9 mm per year for the lingual incisor contact point to -0.2 mm per year for gnathion. Males showed significantly greater rates of vertical growth than females, especially for the upper half of the symphysis. Vertical growth rates were also greater during puberty than during childhood. The horizontal growth changes indicated lingual movement of most symphyseal landmarks. Annual rates of growth were greatest for landmarks located in the upper half of the symphysis. B-point showed the greatest lingual drift. During puberty, the mandibular incisors in females moved lingually as the upper anterior half of the symphysis was remodeled; in males, the incisors maintained their horizontal position while the labial sulcus developed.

Soft Tissue Changes During Adolescence

Soft tissue profile change is a result of varying degrees of skeletal growth and soft tissue thickening. As soft tissue contours between ANS and pogonion are established by age 16, continued projection of the soft tissue profile in a horizontal and vertical direction from age 16 to 20 is the result of underlying skeletal growth, not increased

soft tissue thickness. Nasal tip soft tissue growth is quantitatively the largest parameter noted over the entire age period. Variability of the mean may approach or exceed the actual mean change over all age periods, making clinical comparisons on an individual basis difficult. Continued skeletal and soft tissue movements throughout the 14 to 20-year age period may have significant clinical impact on maintenance of the post treatment profile and post treatment occlusal retention requirements.

The growth of the nose seems to be related to the skeletal growth to a certain extent, but the soft tissue growth is probably responsible for the differences in size between boys and girls. Girls show a decline in nasal growth, whereas boys show an increase in growth velocity after the age of 12 years. At 9 years of age, girls grow very quickly, whereas boys are still growing slowly; at their preadolescent rate. The age at which the growth rate is the same for boys and girls is determined by the intersection of the velocity curves for boys and girls, usually around the age of 12.

The nasolabial angle is larger in girls than in boys and decreases with age more in girls than in boys. The reason for this may be that the tip of the nose is sustained by the nasal septum and the ANS. The ANS is carried forward with age. The A point moves relatively distal with age, and the upper lip grows only slightly in the vertical direction, especially in girls.

The upper lip length showed hardly any increase in the age span studied. In girls, the growth velocity decreased during puberty. The distance between the tip of the incisor and the lowest point of the upper lip, is larger in girls than in boys and increases more in girls than in boys, indicating that girls will have a higher lipline than boys. The distance from the tip of the incisor to the ANS increases with age in both girls and boys as could be expected with the eruption of the incisor. Since the growth velocity of the upper face height decreases very early in girls, the increase in total face height may be attributed mainly to the growth of the lower jaw and the alveolar processes in girls after puberty.

The study by Prah-Anderson and others showed that the girls with a high gumline should be cautiously treated with intrusion because no spontaneous correction can be expected with age, whereas this may be the case in boys. The early (before puberty) decrease in the growth velocity of the upper anterior face height in girls should

also be taken into account when dealing with patients having problems in the vertical dimension.

The upper lip thickness increases with age but the growth velocity decreases during puberty in girls. The lip thickness measured between the subnasale and ANS does not follow this trend exactly because the distance is larger in girls than in boys during puberty. The lip thickness is larger in boys than in girls because point A apparently moves relatively distal.

Regarding the upper lip, sexual dimorphism was demonstrated in the vertical dimension. Furthermore, the position of the upper lip is higher in girls than in boys, in relation to the upper incisor.

Regarding the lower lip, the differences related to gender were mostly found in the horizontal direction. Girls stop growing earlier than boys. The lower lip in boys pouts more than in girls. This cannot be explained by a larger lip thickness (less than 1 mm in boys), but is probably due to a change in lower lip structure.

A thorough knowledge and understanding of the changes in soft tissue during growth is important for the orthodontist. The facial profile responds to changes in the lips and it may be one of the keys to prediction of stability after orthodontic treatment. Functional or orthopedic appliances used in growth modification therapy can be best used in correlation with pubertal growth spurt.

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Growth Studies and Assessment of Age

CHAPTER OUTLINE

- Methods of Growth Measurements
- Craniometry
- Vital Staining
 - Madder feeding
 - Alizarin red S injections
- Histological Method
- Histochemical Studies
- Implants
- Indirect Measurements
- Serial Cephalometric Radiography and Implantation
- Assessment of Age
- Developmental Ages
 - Chronological age
 - Somatotype age
 - Height and weight age
 - Skeletal age
 - Dental age
 - Sexual age
 - Facial age
- Skeletal Age Assessment
 - Carpal bones
 - Different methods of assessment
 - Cervical vertebrae method
 - Mandibular canine calcification method
 - Mandibular third molar
 - Frontal sinus
 - Prediction procedure

The dynamics of the growth of the craniofacial skeleton is a fascinating, complex mechanism. Various methods have been proposed to measure growth which include measurement on living individuals and dry skulls, and indirect measurements taken by means of virtual reproduction of the craniofacial skeleton. Essentially, the various study methods used to assess growth, try to find out answers to the following—pattern of growth, sites

of growth, amount and rate of growth, direction and factors influencing growth.

METHODS OF GROWTH MEASUREMENTS

Proffit has classified the methods used to study growth as (Flow chart 10.1) (i) Measurement approaches and (ii) Experimental approaches.

Measurement approaches are based on techniques used for measuring living animals and these methods do not harm the animal or human beings. They can be used for longitudinal studies which means that the animal will be available for subsequent measurements. The various measurement approaches include:

- Craniometry
- Anthropometry
- Cephalometric radiology
- Three dimensional imaging.

Experimental approaches: This procedure is invasive and may cause harm to the animals. It is done only on animals and is used for cross sectional studies. The following are the various experimental approaches:

- Vital staining
- Radioactive tracer
- Autoradiography
- Implant radiography.

Sarnat in 1986 has classified the growth measurement methods as (i) Direct measurements and (ii) Indirect measurements.

Direct measurements:

- Anthropometry
- Vital staining
 - i. Madder feeding
 - ii. Alizarin red S injection

madder. This can be obtained by a single intraperitoneal or intravenous injection of a 2 percent solution. Ground sections which are 25 to 50 μm thick are prepared for the microscopic viewing of staining effects. Sharp red lines were seen under the dissecting microscope under reflected light. Under higher magnification and strong illumination, the red lines (5-20 μm in width) are readily counted and the distance between them can be accurately measured with a micrometer eyepiece. A method for obtaining 100 μm serial sections of undecalcified bones, including the skull, has also been used with considerable success (Hoyte, 1968).

Advantage

At one injection of the dye, several red lines can be found. This can be the result of either deposition of the dye at the same time in several areas of active calcification or the improper plane of section of a bone.

Disadvantage

As resorption may lead to the removal of stained bone, vital staining will give incomplete data on the pattern of bone formation.

Other dyes used for vital staining include procion, tetracycline, fluorochrome, lead acetate, trypan blue, and sodium fluoride.

Although studies using vital stains are not possible in humans, vital staining can occur inadvertently. Many children born in the late 1950's and the early 1960's were treated with tetracycline. It was discovered that tetracycline is an excellent vital stain that binds to calcium at growth sites in the same way as alizarin. However, it poses esthetic problems like discoloration of teeth when the drug is administered during mineralization of teeth.

HISTOLOGICAL METHOD

Histological studies are primarily qualitative in nature and are used to elucidate processes responsible for growth. The problems involved in bone formation are of three types:

- Origin and transformation of bone cells from the undifferentiated mesenchyme;
- The formation and composition of the matrix substances; and
- Mode of deposition of the bone salts in the matrix.

Bone tissue is in a state of continuous change due to interplay of apposition and resorption. Osteoblasts are arranged like a cuboidal epithelium in single layer of cuboidal cells on the surface of the bone. Osteoclasts are found in areas where bone is being destroyed or resorbed. The osteoclasts, are multinucleated giant cells of varying size and shape and are found in shallow hollows (Howship's lacunae) on the surface of the bone trabeculae. Bhaskar (1953) compared the prenatal and postnatal growth, and development of the mandible in normal rats and in those characterized by retardation of bone resorption.

Enlow (1990), on the basis of extensive histologic studies, reconstructed gross patterns of bone formation and destruction.

HISTOCHEMICAL STUDIES

Histochemical studies are valuable in obtaining further information about the nature of bone formation. By this method, for instance, the importance and the localization of enzymes (alkaline and acid phosphatase and other substances like glycogen and glycoprotein) are studied.

Implants

Duhamel (1742) introduced implants in the study of growth of bones. He was followed by Hunter who inserted two pellets in the shaft of the tarsus of a young pig and measured the distance between the pellets. The distance remained same after the tarsus was grown, suggesting no interstitial growth in bone. Spoylar et al evaluated the stability of implants. It was found that implantation of gold, silver, dental silver amalgam, stainless steel, vitallium, and tantalum in the form of screw, pegs, pins, clips, or wires within a single bone can be used for the study of total amount of bone growth by measuring the increase in distance between the implants and the outer borders of the bone (Sarnat 1968, Sarnat and Selman 1978). Humphrey (1863) placed wire loops around the ramus of pig mandible, and demonstrated resorption on anterior border of ramus and deposition on the posterior border. Serial data is not provided by this method without reoperation or killing the animal. This method has also been used to determine sutural growth by placing implants on either side of the suture. The historical review of implant markers is given in Table 10.1.

Table 10.1: Brief historic review of implant markers used in the longitudinal study of the growth of bones

Investigator	Year	Material used	Bones studied	Animal
<i>Gross (direct) studies</i>				
Hales	1727	Holes	Tibia	Chicken
Duhamel	1742	Silver stylets	Long bone	Pigeon, dog
Hunter	1770	Lead shot	Tibia, tarso-metatarsal	Pig, chicken
Humphry	1864	Wires	Mandible	Pig
Gudden	1874	Holes	Parietal, frontal	Rabbit
Wolff	1885	Metal	Frontal, nasal	Rabbit
Giblin and Alley	1942	Wax	Parietal, frontal	Dog
Roy and Sarnat	1956	Stainless steel wire, black silk suture	Rib	Rabbit
<i>Gross (direct) and/or serial roentgenographic (indirect) studies</i>				
Dubreuil	1913	Metal	Tibia	Rabbit
Gatewood and Mullen	1927	Shot	Femur	Rabbit
Troitzky	1932	Silver wires	Skull	Dog
Levine	1948	Dental silver amalgam	Frontal, nasal	Rabbit
Gans and Sarnat	1951	Dental silver amalgam	Various facial	Monkey
Sissons	1953	Metal	Femur	Rabbit
Selman and Sarnat	1953	Dental silver amalgam	Frontal, nasal	Rabbit
Robinson and Sarnat	1955	Dental silver amalgam	Mandible	Pig
Bjork	1955	Tantalum	Various facial	Human
Elgoyhen and associates	1972	Tantalum	Various facial	Monkey
Sarnat and Selman	1978	Dental silver amalgam	Nasal	Rabbit
Sarnat and McNabb	1981	Tantalum	Plastron	Turtle

INDIRECT MEASUREMENTS

- *Impressions and study casts:* Duplication of various parts of the body and extremities is possible by taking impressions with the plaster of Paris, hydrocolloid, thoiokol rubber, low fusing metal, stone or other material. (Sarnat et al 1953). Individual or sectional impressions are taken for the particular part which is to be duplicated. The impression serves as the negative, and by filling it with plaster of Paris, an accurate duplicate is obtained, which serves as a record and can be compared with models made at a later stage of growth and development.
 - *Photographs:* The effect of disease on the face jaws, teeth, and the human constitution have been shown in photographs. Photographs taken under controlled conditions with the subjects placed against a graduated grid have permitted morphologic classification. Sheldon, Stevens, and Tucker used such grids for establishing body types. This method does not lend itself to accurate measurements of growth of individual bones, but it does permit the study of growth of selected regions or the entire subject.
3. *Radio autographs:* Radio autographs are obtained by injecting radioactive isotopes and by placing a

photographic emulsion for suitable exposure period in close contact. Alpha or beta rays emitted from the radioactive material affect the silver bromide crystals on the photographic emulsion in a manner similar to that of light. After development, dark areas correspond to distribution of radioactive material (Bartelstone, 1950). Many substances were used for autoradiography, which include sodium, calcium, strontium, fluorine, chlorine, iodine, plutonium, uranium, americium, and gallium. Now radioactive isotope ^{99m}Tc can be used to detect areas of rapid bone growth. This method is more useful in the diagnosis of localized growth problems than in studies of growth pattern. Studies in autoradiography in bone and cartilage have been done by Long et al in 1968 and Gross et al in 1951. Dixon and Hoyte (1963) compared the autoradiographic and alizarin techniques in the study of bone growth.

- *Radiographs:* Radiography is a reliable method of studying growth of bones. In 1912, Tandler suggested the use of X-ray films in the studies of anthropometry of the skull. In 1931, Broadbent and Hofrath, simultaneously but independently, described a technique of cephalometric radiography. In 1937,

Broadbent described the findings from his studies on growing children. This was a cross-sectional method but by serial super positioning with serial radiographic tracings on stable bony landmarks provided longitudinal data. In 1941, Brodie was the first to apply Broadbent's method to a longitudinal growth study of human males from the third month to the eighth year of life. The accuracy of the method depends on standardization of technique. However, selection of a stable anatomic base, for superimposing the radiographic tracings is the key to reliable findings.

Advantages

- This method eliminates serious deficiencies of anthropologic techniques.
- It permits a dynamic study of the growing child, i.e. increase in size and change in proportion of the same growing bone or group of bones forming a bone complex (as in the middle third of the face and the neurocranium).
- It reveals rate, amount and relative direction of bone growth.

Disadvantages

- In this technique three-dimensional information is being interpreted as a two-dimensional process.
- In addition (Moyers and Bookstein 1979), the conventional cephalometric fails to capture the curving of form and its changes and thus misrepresent growth.
- Radiation exposure.

SERIAL CEPHALOMETRIC RADIOGRAPHY AND IMPLANTATION

Serial radiography with radio opaque implants is a more accurate and reliable approach for a dynamic longitudinal study of the growth of bone(s). Robinson and Sarnat in 1955 used this method in growth study of the mandible in the pig, McNamara and Graber (1975) and Bjork (1963) used it in humans. Bjork in 1968 and Bjork et al in 1983 have studied about the growth rotations of the mandible using implant radiography.

Advantages

- Increase in size and the change in proportion can be recorded.

- A stable base for superpositioning the serial radiographic tracings is obtained by inserting two or more radio-opaque implants. Thus, growth can be accurately predicted by superpositioning the tracings over the implants.

The measurements are valid only if the implants do not extrude into the surrounding soft tissues and foreshortening of implants must be avoided for which the implant must lie parallel to the X-ray film.

Other Considerations

Teeth, alone or in combination with other methods, serve as accurate, permanent chronologic recorders of systemic conditions. Added to the above methods are digital subtraction radiography, computed axial tomography, magnetic resonance imaging and sonic interferometry, biorthogonal grids, elliptical Fourier functions and finite elements (Melvin Moss). Computed axial tomography allows three-dimensional construction of the face, but produces significant radiation exposure. However, cone beam computerized tomography has reduced the radiation exposure significantly. Superimposition of 3-D images is more difficult than for the 2-D images, but methods developed recently help in overcoming this difficulty. Magnetic resonance imaging is another method which has the advantage of no radiation exposure. This method has been applied to analyze growth changes after functional appliances. The approximate information provided by the different methods is given in Table 10.2.

ASSESSMENT OF AGE

Growth assessment is of primary concern in planning orthopedic correction and surgical treatment of skeletal malocclusion. Estimation of growth potential requires the assessment of the developmental age of the individual patient.

Developmental age is being classified by Krogman into five types (Flow chart 10.2):

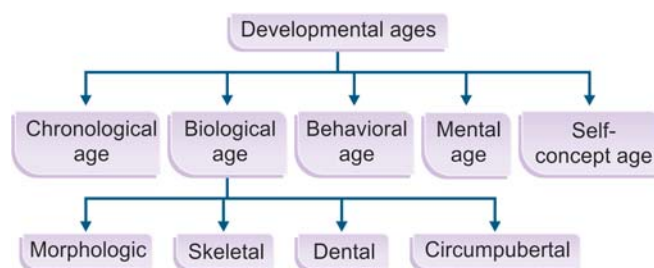
- Chronological age
- Biological age which consists of morphologic age-skeletal age, dental age and circumpubertal age
- Behavioral age
- Mental age
- Self-concept age.

Table 10.2: Approximate information provided by various methods used to study postnatal bone growth—modified from Sarnat (1984)

Method	Growth				Type of study	Limitations
	Site	Amount	Rate	Direction		
Osteometry						
Skeletal remains	0	X	X	X	Cross-sectional	Material of unknown history, posthumous distortion.
Living	0	X	X	X	Longitudinal	Soft tissues restrict accurate measurement.
Vital staining ²	XXX	XXX	XX	X	Longitudinal	Toxicity, method requires refinement.
Implant markers ^{1,2}	XX	XXX	X	X	Longitudinal	Local reaction to implants, requires reoperation.
Histological and histochemical methods	XXXX	X	0	X	Cross-sectional	Sections show conditions at time of death.
Impression and casts	0	XX	XX	XX	Longitudinal	Soft tissue restrict accuracy of impression.
Photographs	0	X	X	X	Longitudinal	Two-dimensional study of three dimensional process.
Serial radiographs	0	XXX	XX	XX	Longitudinal	Must obtain stable landmarks, three-dimensional information not accurate, radiation exposure.
Serial radiography and implantation ^{1,2}	XXX	XXX	XXX	XXX	Longitudinal	Three dimensional information not entirely accurate, radiation exposure.
Serial radiography and metaphyseal bands ³	XXX	XXX	XXX	XXX	Longitudinal	Record of a toxic process, rate of growth not normal.
Radioautographs	XXX	0	0	0	Cross-sectional	Primarily of qualitative value.

0—Gives no information, X shows trends, XX shows relatively accurate, XXX grossly accurate; XXXX microscopically accurate.
 1—Sutural growth
 2—Apposition and resorption
 3—Endochondral growth

Flow chart 10.2: Different types of developmental ages



Chronological Age

Chronological age is the measurement by the period of time (years and months) for which something or someone has existed. It is the most easily determined parameter of all the developmental ages, which can be easily figured out from the child's date of birth. The

number of years a person has lived, used especially in psychometrics as a standard against which certain variables such as behavior and intelligence are measured, is called the chronological age. Each child has his or her own characteristic time clock. Some are early maturers, some others late maturer, while the rest fall in the middle. Factors like disease, metabolic disturbances and endocrine disorders, and environmental affect the maturation of the child. Hence, there is a lack of correlation between the developmental age and chronological age. Therefore, it is a poor indicator of developmental status.

Somatotypic Age

Sheldon has divided somatotypes into three categories, namely ectomorph, mesomorph, and endomorph. The somatotype is defined by a series of 17 anthropometric

measurements and is related to nutritional status. The endomorph is stocky, has abundant subcutaneous fat, and has digestive viscera that are highly developed; somatic structures are relatively underdeveloped. The mesomorph is upright, sturdy, and athletic in whom the muscles, bones and connective tissues predominate. The ectomorph is tall, thin and fragile. His extremities are long and slender with minimal subcutaneous fat and muscle tissue. In general, the ectomorph is a late maturer (adolescent spurt occurs one year after the mesomorph) whereas the endomorph is an early maturer in terms of chronological age. Somatotype gives an idea of the developmental status, but it is not an accurate predictor.

Height and Weight Age

Height and weight age can be used as a parameter for assessment of developmental age. The standard growth curve in which the child's height is compared with children of same chronological age is used to assess height age from two years up to the onset of puberty after which there is variation due to the differences in the timing of puberty. The child's height is plotted in a curve which is designed for his race and the age is calculated on the 50th percentile isobar. As the child's height is being influenced by genetic and environmental factors as well as chronological age, his height is of limited value in assessing developmental status.

Another important measurement is growth velocity curve in which height increments are plotted for each year. There is a short prepubertal growth peak between the ages of 6 and 7 which is not uniformly present; a pre-pubertal minimum, acceleration at the pubertal peak which averages 11.5 years in females, 14.5 years in males and finally a deceleration until adult height is attained. [The shorter the duration of the height spurt, the more intense it will be. The longer the duration of the height spurt, the less intense it will be (e.g. late maturers)]. Male growth spurt is later and more intense than the female which accounts for the sex difference in adult height.

Likewise weight curves are also constructed. Though it is obvious that weight curves correlate with height, it is clear that abnormal variation in weight in otherwise normal children limits the usefulness of these curves as a sole indicator of developmental age.

Skeletal Age

Skeletal maturation has been used to assess a child's developmental age. Bones from different parts of the

body were used to assess the skeletal age. More commonly the hand wrist radiographs are used. A characteristic pattern of ossification of the hand wrist bones was found and correlated with the development. The union of the epiphyses with their diaphysis occurs in a specific order, which in females is advanced by 3 to 4 years compared to males. Between 12.5 and 14 years, the most active transformation of the epiphyseal cartilages occurs concurrently with the peak height velocity. Skeletal age was found to more highly correlate with menarcheal age than with height, weight or annual increments in height, and menarche usually occurred soon after the fusion of the epiphysis of the distal phalanges with their shafts.

Dental Age

Numerous methods have been proposed to assess dental age. Dental age does not correlate well with the developmental age. The dental age method involves the recognition of clinically present teeth with eruption charts. The major limitation in this method is the variation in the timing of eruption, the influence of local and environmental factors, and the fact that several or no teeth may erupt during the same time interval.

Nanda, in his study, found a poor correlation when he compared age at which all permanent teeth are present and the circum-pubertal growth spurt. Bambha and Natta in their study of 60 children, found no evidence of association between the time of tooth eruption and the time of skeletal maturation.

Demisch and Wartman found a high correlation between calcification of the mandibular third molar and skeletal and chronological age ($r = 0.73$, and 0.86). However, the skeletal age and chronological age are well correlated ($r = 0.89$ and 0.92). Demirjian et al proposed a method of determining dental age by scoring the stage of calcification of seven teeth on the left side of the mandible and the construction of dental maturity standard curves. Each tooth is given a point value according to this state of development. The sum of the individual points gives the development value, which can be transferred into the dental age with the aid of standard tables. The smaller the sum of points, the younger the dental age; the higher the sum, the older is the dental age.

Nine relevant stages of dental development:

- 0 - Tooth germ without signs of calcification.
- A - Calcification of single occlusal points without fusion of different calcifications.

- B - Fusion of mineralization points; the contour of the occlusal surface is recognizable.
- C - Calcification of the crown is complete beginning of dentin deposits.
- D - Crown formation is complete up to the cemento-enamel junction.
- E - Root length shorter than crown height.
- F - Root length larger than crown height.
- G - Root formation is finished. Apical foramen is still opened.
- H - Apical foramen is still closed.

Sexual Age

During the time of puberty, various hormones yield characteristic body changes. The stages of secondary sexual characteristics provide a physiological calendar of adolescence that correlates with the individual's physical growth status. Adolescence in girls can be divided into three stages, based on the extent of sexual development. The first stage occurs at about the beginning of the physical growth spurt (appearance of the breast buds, early stages of pubic hair development), and stage II after one year of stage I during which peak velocity of growth occurs. The third stage, 1 to 1½ years after stage II, is marked by the onset of menstruation (menarche).

The stages of sexual development in boys are very difficult to specify; starting with stage I characterized by "fat spurt" which is marked by gain in body weight and an increase in the size of the scrotum. Stage II begins one year after stage I and marks the beginning of the height spurt, followed by stage III (8-12 months) marked by peak velocity in body height. Stage IV which occurs 15 to 24 months after stage III is difficult to pinpoint and is marked by the end of spurt of growth in height.

Facial Age

The aim of the developmental age assessment is for orthopedic or functional intervention of skeletal malocclusions. The objective is to identify where the children, are on their facial growth curve, and to use this as the predictor for future growth. The methods used were anthropometric measurements and development of facial growth velocity curve using measurements from serial cephalometric radiographs similar to the standard height curves. It is very difficult to develop growth curves from serial cephalometric radiographs. Therefore, it is best to find parameters correlating to facial age. Nanda

in one of his studies measured seven linear measurements—S-Gn, N-Gn, S-Go, Go-Gn, S-N, N-Pr and Id-Gn. Each of the measurements and the yearly measurements were plotted on a graph versus chronological age. These curves have the same basic form as the standard height curves; S-Gn and N-Gn were most like the other skeletal growth curves; S-N appeared to be a composite of both skeletal and neural growth; N-Pr and Id-Gn correlated with the emergence of the permanent teeth. The final body height reached before the peak in facial growth. Bergersen in his study stated that initiation of the growth spurt in height and enlargement of the face occurred at an average skeletal age of 12.5 years as determined by hand wrist radiographs and this correlated with the appearance of sesamoid bone. Bjork found that completion of upper facial height occurs at DP3 U stage. Tofani in his study, showed that mandibular growth of females during puberty exhibited a peak 10 months before menarche in early maturing females and 5 months after menarche in late maturing females. Hunter showed that 50 percent of the maximal facial increments occurred at the same time as maximum growth in height and only 29 percent occurred after the maximum.

SKELETAL AGE ASSESSMENT

Bone age: Bone age is an indication of physical development and maturation of the skeleton. Standards obtained by means of roentgenograms are employed to determine the order, rate, time of appearance, and progress of ossification of various centers of skeletal ossification.

Bone age can be calculated by the absence or presence of various osseous centers in several regions of the body and compared with the standards. Bones such as carpals, femur, the elbow joint, the shoulder joint and the skull can be used for this purpose. The hand and the wrist bones which present numerous secondary ossification centers from birth are commonly used.

Carpal Bones

Carpal bones (Fig. 10.1) were first named by Lyser in 1683. Each carpal bone except the pisiform has six surfaces: proximal, distal, volar, dorsal, lateral and medial.

The carpal bones include the proximal and distal rows (Flow chart 10.3).

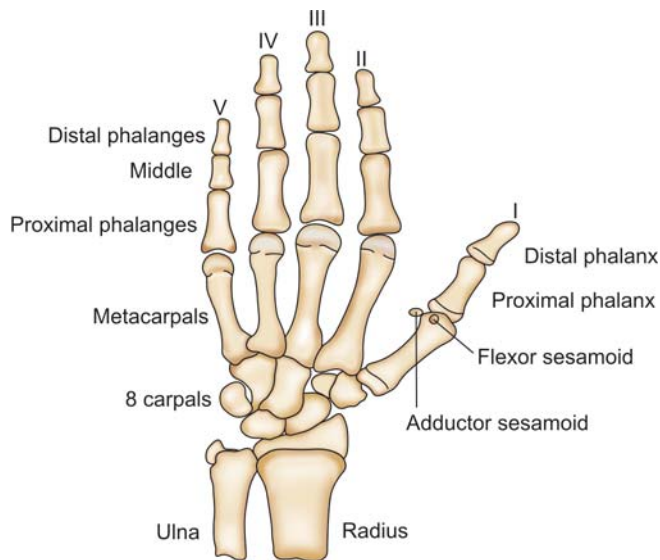
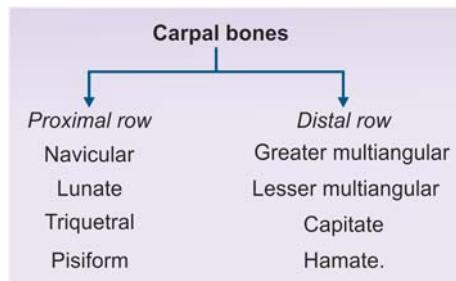


Fig. 10.1: Carpal bones

Flow chart 10.3: Carpal bones



Advantages of using carpal bones in assessing skeletal maturation:

- Carpal bones, epiphysis and phalanges provide a clue to bone growth in the body as a whole.
- Ossification occurs in the period after birth and before maturity.
- The bones are easily accessible. Clothing need not be removed.
- Less time consuming technique.

Indications of Hand wrist radiographs

- Prior to rapid maxillary expansion.
- When maxillomandibular expansion changes are indicated in the treatment of class III cases, skeletal class II or skeletal open bites.
- In patients with marked discrepancy between dental and chronological age.

- Orthodontic patients requiring orthognathic surgery if undertaken between the ages of 16 and 20 years.
- For planning of growth modification treatment.

History

Ranke (1896) is considered to have been the first to study skeletal development progress by means of wrist roentgenograms. *Rotch (1910)* recognized that weight, height, and tooth eruption were at best only rough estimates of physical maturity in terms of chronological age. *Bayley* found a correlation between maturation of the knee as seen on the roentgenogram up to 13 years. *Garn and Rohmann* concluded that hand-wrist ossification is useful in detecting growth abnormalities but it is not a precise method of measuring developmental progress in normal individuals.

Todd TW (1931) started a longitudinal study by taking a series of periodic hand and wrist radiographs of growing children in Cleveland, Ohio USA. Unfortunately, Professor Todd died in 1938 after publishing the initial data of his study in 1937. After his death, the study was continued and *William Greulich and Idell Pyle* compiled the *Radiographic atlas of skeletal development of hand and wrist*, which was published in 1950 and revised in 1959. The atlas contains standards, which were developed on the basis of skeletal age as opposed to chronological age.

Greulich and Pyle's radiographic assessment of the hand-wrist complex involved two specific steps, the atlas and the bone specific methods. The atlas method involved comparing a hand-wrist film with the standard of the same sex and nearest chronological age. The film would then be compared with adjacent standards, both older and younger than the one which is of the nearest chronological age. Finally, the standard which appears most closely to resemble the film in question is chosen. This first method is termed the *atlas method* (Greulich and Pyle, 1959).

After selecting the appropriate GP2 standard via the atlas method, the examiner should proceed to make a more detailed comparison of the individual bones and epiphyses visible in them. The bones of the hand-wrist complex should be considered in a regular order. That is, one should begin at the distal ends of the radius and ulna, proceed next to the carpals, then to the metacarpals, and finally to the phalanges. Similarly, one should

examine the carpals in a regular sequence—in their usual order: Capitate, Hamate, Triquetral, Lunate, Scaphoid, Trapezium, Trapezoid, Pisiform. This method is termed the *bone specific method* (Greulich and Pyle 1959). Each center is given a skeletal age either that of the standard or that of the one before or after corresponding exactly with its developmental status. An overall age is then determined.

Tanner and Whitehouse Method

Tanner and Whitehouse suggested three methods of scoring maturity of individual bones to determine skeletal age.

Radius, ulna, short (RUS) bones score, rates the radius, ulna, metacarpals of digits, 1, 3, and 5, middle phalanges of digits 3 and 5, and distal phalanges, of digits 3 and 5.

The carpal bone method scores capitate, hamate, triquetral, lunate, scaphoid, trapezium and trapezoid. The problem of using the carpal bones is that only 97 percent of the carpal score is reached by age 13 in males and age 11 in females.

The TW2 method does not use a scale based on the age, rather it is based on a set of bones' standard maturity for each age population. In the TW2 method, twenty regions of interest (ROI's) located in the main bones are considered for the bone age evaluation. Each ROI is divided in three parts: epiphysis, metaphysis and diaphysis especially in young people, it is possible to identify these different ossification centers in the phalanx proximity (Fig. 10.2). The development of each ROI is divided into discrete stages and each stage is given a letter (A, B, C, D... H) (Fig. 10.3). A numerical score is associated with each stage for each bone (Table 10.3). By adding the scores of all ROIs, an overall maturity score is obtained.

Bjork, Grave and Brown Method (1976)

Figure 10.4 shows the nine developmental stages with the ossification events localized in the area of the phalanges, carpal bones and radius. The developmental stages are assessed according to the relation between the epiphyses and the diaphyses. There are three stages of ossification of the phalanges:

First stage: Epiphyses show the same width as the diaphysis in this stage.

Second stage: Capping stage—epiphysis surrounds the diaphysis like a cap.

Third stage: U-stage: bony fusion of the epiphysis and the diaphysis.

For the assessment of the maturity in the area of phalanges, fingers 1 to 5 beginning with the thumb are labeled.

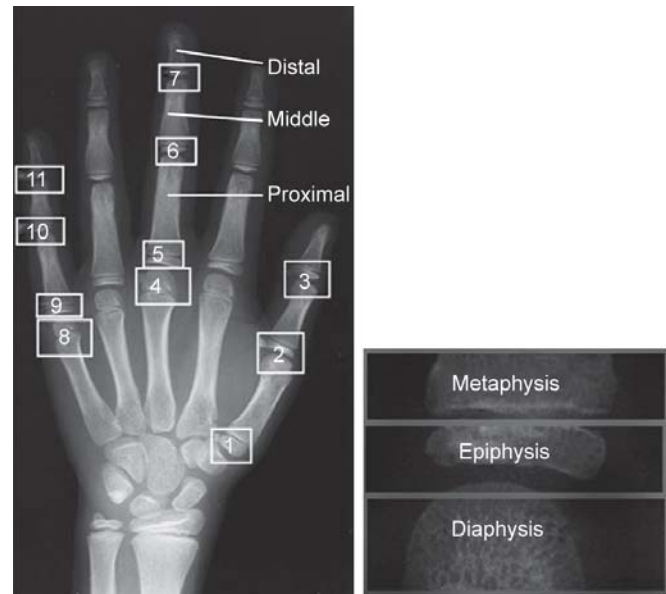
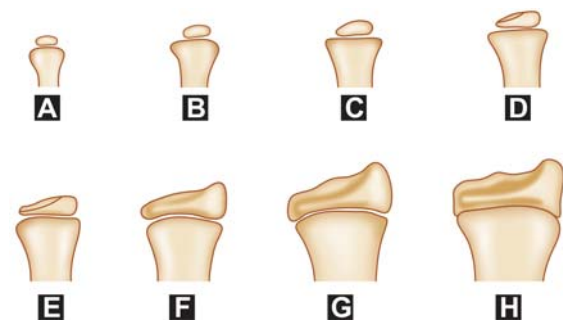


Fig. 10.2: Parts of carpal bones—radiographic assessment in regions of interest. (1) First metacarpal; (2) Proximal phalanx of the thumb; (3) Distal phalanx of the thumb; (4) Third metacarpal; (5) Proximal phalanx of the third finger; (6) Middle phalanx of the third finger; (7) Distal phalanx of the third finger; (8) Fifth metacarpal; (9) Proximal phalanx of the fifth finger; (10) Middle phalanx of the fifth finger; (11) Distal phalanx of the fifth finger.



Figs 10.3A to H: Discrete developmental stages of TW2 method

Table 10.3: Numerical scores for each bone—TW2 method of Tanner and Whitehouse

	Numerical score for each bone															
	Female stage								Male stage							
	B	C	D	E	F	G	H	I	B	C	D	E	F	G	H	I
<i>TW2 bones</i>																
Radio	17	19	25	33	54	85	99	106	15	17	21	27	48	77	96	106
Ulna	22	26	30	39	60	73	80	0	22	26	30	39	56	73	84	0
Metac. I	5	6	11	18	24	29	31	33	4	5	11	19	24	28	30	32
Metac. III	3	5	7	11	17	23	24	26	3	4	6	10	16	22	23	25
Metac. V	3	4	7	12	18	22	24	25	3	3	6	12	17	21	23	25
Fal. Prox. I	5	5	8	14	24	29	30	32	4	5	8	15	23	28	30	32
Fal. Prox. III	4	4	7	13	20	24	25	26	3	4	6	13	20	23	24	26
Fal. Prox. V	4	4	7	13	19	23	24	25	3	3	6	13	19	22	23	25
Fal. Media III	4	4	7	13	20	23	24	25	3	4	7	13	19	22	23	25
Fal. Media V	4	5	8	14	20	22	22	23	4	4	8	14	19	21	22	23
Fal. Distale I	5	5	8	15	24	31	32	34	4	4	7	14	23	30	31	33
Fal. Dist. III	3	4	6	10	17	22	23	24	3	4	6	10	16	21	22	24
Fal. Distale V	3	4	7	11	17	21	22	23	3	4	7	11	16	20	21	23
Capitato	53	56	61	67	76	85	113	0	60	62	65	71	79	89	116	0
Uncinato	44	47	53	64	74	85	97	109	42	44	49	59	70	81	92	106
Piramidale	8	12	19	28	36	46	63	0	7	10	17	28	38	45	62	0
Semilunare	10	14	20	27	35	46	60	0	10	13	20	27	36	44	60	0
Scafoide	13	17	23	29	36	44	57	0	14	18	23	30	35	42	58	0
Trapezio	12	14	20	25	32	39	49	59	12	15	21	28	34	39	47	59
Trapezoide	13	16	20	24	31	40	57	0	14	16	20	23	32	39	56	0

*First Stage of Maturation**PP2 stage*

The epiphysis of the proximal phalanx of the index finger (PP2) has the same width as the diaphysis. This stage approximately occurs 3 years before the peak of the pubertal growth spurt.

*Second stage**MP3 stage*

Epiphysis of the middle phalanx of the third finger (MP3) is of the same width of the epiphysis.

*Third stage**Pisi, H1 and R stage*

This stage of development can be identified by three distinct ossification areas; these show individual variations but appear at the same time during the process of maturation.

Pisi stage = visible ossification of the pisiform.

H1 stage = ossification of the hamular process of the hamatum.

R stage = same width of the epiphysis and the diaphysis.

Fourth stage: S and H2 stage

S stage = first mineralization of the ulnar sesamoid bone of the metacarpophalangeal joint of the thumb.

H2 stage = progressive ossification of the hamular process of the hamatum.

The fourth stage is reached shortly before or at the beginning of the pubertal growth spurt.

Fifth stage: MP3cap, PP1cap and R cap stage

During this stage, the diaphysis is covered by the cap shaped epiphysis.

In the MP3 cap stage, the process of ossification begins at the middle phalanx of the third finger.

In the PP1 cap stage, at the proximal phalanx of the thumb and

In the R cap stage at the radius.

This stage of ossification marks the peak of the pubertal growth spurt.

Sixth stage: DP3u stage

Visible union of epiphysis and diaphysis at the distal phalanx of the middle finger (DP3).

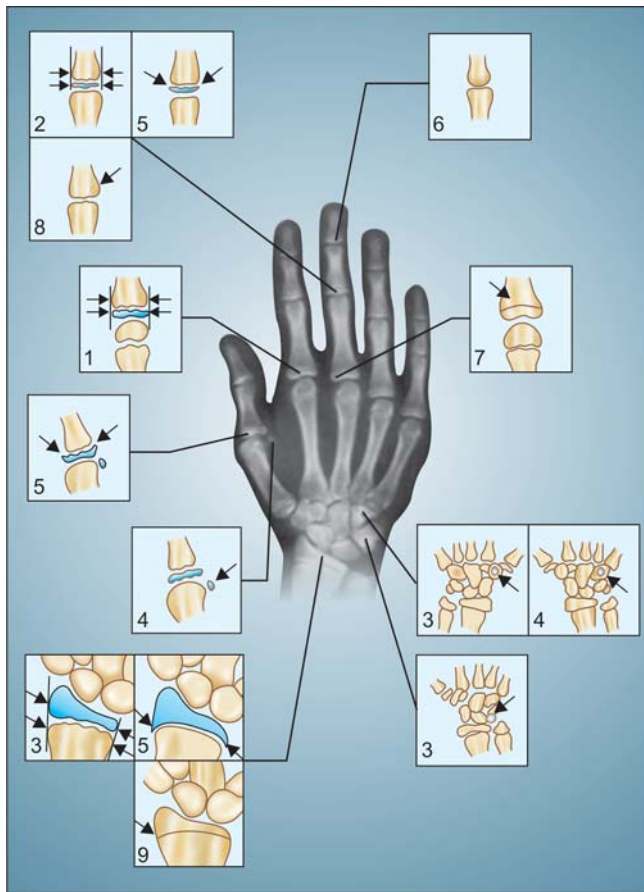


Fig. 10.4: Bjork, Grave and Brown method of assessment of hand wrist X-rays

This stage of development constitutes the end of pubertal growth.

Seventh stage: PP3u stage

Visible union of the epiphysis and diaphysis at the proximal phalanx of the little finger (PP3).

Eighth stage: MP3u stage

Union of epiphysis and diaphysis at the middle phalanx of the middle finger is clearly visible (MP3).

Ninth Stage: Ru stage

Complete union of the epiphysis and diaphysis of the radius.

Singer's Method of Skeletal Maturity Assessment

This method of assessment was proposed by Julius Singer in the year 1980. Six stages of hand wrist development are described.

Stage one: This stage is characterized by absence of the pisiform, absence of hook of hamate, with the epiphysis of proximal phalanx of second finger being narrower than its diaphysis.

Stage two (pre pubertal): Stage two is characterized by initial ossification of hook of the hamate, initial ossification of the pisiform and proximal phalanx of the second finger being equal to its epiphysis. Stage two represents that period prior to the adolescent growth spurt during which significant amounts of mandibular growth are possible. Maxillary orthodontic therapy in conjunction with mandibular growth might aid correction of a class II relationship with considerable speed and ease.

Stage three (pubertal onset): This stage is characterized by the beginning of calcification of ulnar sesamoid, increased width of epiphysis of proximal phalanx of the second finger and increased calcification of hook of hamate and pisiform.

Stage four: Stage four (pubertal) is characterized by the presence of calcified ulnar sesamoid and capping of the diaphysis of the middle phalanx of the third finger by its epiphysis.

Stage five (pubertal deceleration): This stage is characterized by fully calcified ulnar sesamoid, fusion of epiphysis of distal phalanx of third finger with its shaft, and epiphyses of radius and ulna not fully fused with respective shafts. Stage 5 represents that period of growth when orthodontic treatment might be completed and the patient is in retention therapy.

Stage six (Growth completion): No remaining growth sites are seen.

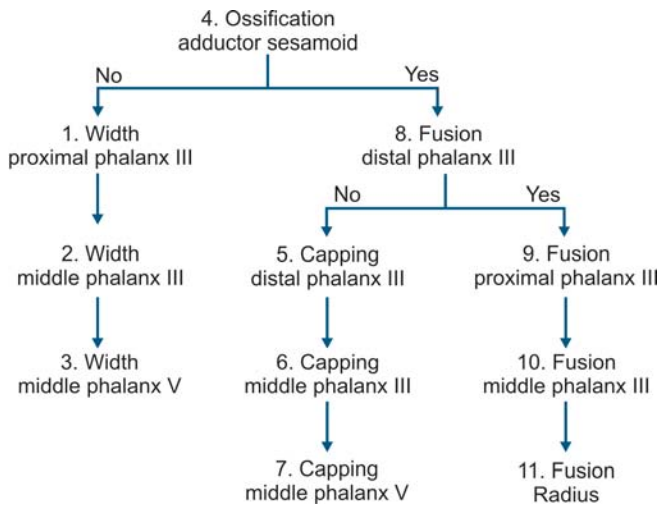
Fishman Skeletal Maturity Indicator

Leonard S Fishman, in 1982 developed this method to predict the skeletal maturation (Flow chart 10.4): It makes use of anatomical sites located on thumb, third finger, fifth finger and radius.

Eleven discrete adolescent skeletal maturity indicators covering the entire period of adolescent development have been described. The Fishman's system of interpretation uses four stages of bone maturation. They are:

- Epiphysis equal in width to diaphysis.
- Appearance of adductor sesamoid of the thumb.
- Capping of epiphysis.
- Fusion of epiphysis.

Flow chart 10.4: Fishman's 11-grade scheme used to assess skeletal maturity from a hand-wrist radiograph



The eleven skeletal maturity indicators are:

SMI 1: The third finger proximal phalanx shows epiphysis and diaphysis of equal width.

SMI 2: Width of the epiphysis equal to that of diaphysis in the middle phalanx of third finger.

SMI 3: Width of the epiphysis is equal to that of the diaphysis in the middle phalanx of fifth finger.

SMI 4: Appearance of adductor sesamoid of the thumb.

SMI 5: Capping of epiphysis seen in the distal phalanx of third finger.

SMI 6: Capping of epiphysis seen in the middle phalanx of third finger.

SMI 7: Capping of epiphysis seen in the middle phalanx of fifth finger.

SMI 8: Fusion of epiphysis and diaphysis in the distal phalanx of the third finger.

SMI 9: Fusion of epiphysis and diaphysis in the proximal phalanx of third finger.

SMI 10: Fusion of epiphysis and diaphysis in the middle phalanx of the third finger.

SMI 11: Fusion of epiphysis and diaphysis seen in the radius.

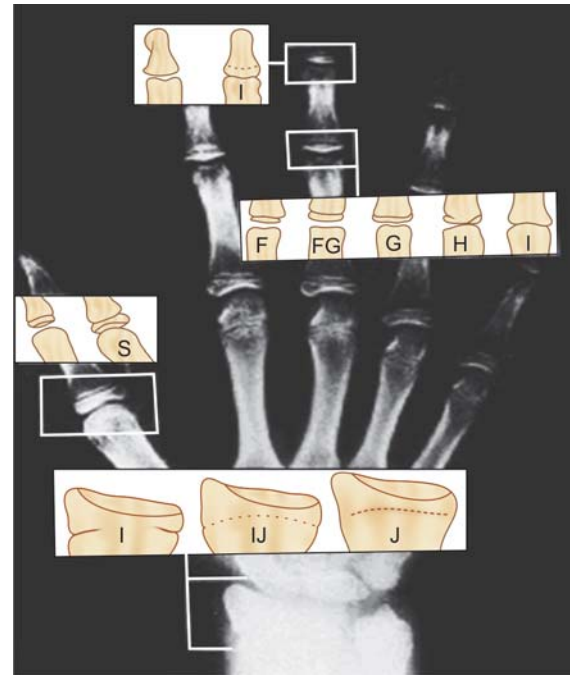


Fig. 10.5: Hagg and Taranger method

Skeletal Maturity Indication Method Developed by Hagg and Taranger (Fig. 10.5)

The assessment was done by taking into consideration the ossification of the ulnar sesamoid of metacarpophalangeal joint of the first finger (S) and certain specified stages of three epiphyseal bones: the middle and distal phalanges and third finger (MP3 and DP3) and the distal epiphysis of the radius (R). Hand wrist radiographs used to develop this method were taken from 6 to 18 years of age.

Sesamoid: Appearance of the ulnar sesamoid is seen during the acceleration period of pubertal growth spurt. (onset of PHV) in 86 percent of girls and 92 percent of boys.

Middle Third Phalanx

- MP3 F:** The epiphysis is as wide as the metaphysis. MP3-F was attained before pubertal onset by about 40 percent of the subjects and at PHV by the rest of the subjects.
- MP3 FG:** The epiphysis is as wide as the metaphysis and there is a distinct medial and or lateral border

of the epiphysis forming a line of demarcation at right angles to the distal border. This stage is attained one year before or at PHV.

- **MP3 G:** The sides of the epiphysis have thickened and also cap its metaphysis, forming an edge distally at one or both sides. This stage is attained at or one year after the PHV.
- **MP3 H:** Fusion of the epiphysis and metaphysis has begun and is attained after PHV but before the end of growth spurt by practically all the boys and about 90 percent of girls.
- **MP3 I:** Fusion of the epiphysis and metaphysis is completed. MP3 I was attained before or at end of pubertal growth spurt in all the subjects except a few girls.

Distal Third Phalanx

DP3 I: the fusion of epiphysis and diaphysis is completed. This stage was attained during the deceleration period of the pubertal growth spurt (PHV-END) by all subjects.

The distal epiphysis of the radius (R):

- Stage I—fusion of the epiphysis and metaphysis has begun.
- Stage IJ—fusion is almost completed but there is still a small gap at one or both margins.
- Stage J—fusion of the epiphysis and metaphysis is completed.
- R-I is attained 1 year before or at end of the pubertal growth spurt by about 80 percent of the girls and about 90 percent of the boys.

R-IJ and R-J are not attained before the end of the pubertal growth spurt by any subject.

Modified Hagg and Taranger by Rajagopal et al (2002) (Fig. 10.6)

Additional bone stage between MP3-H (deceleration of the curve of the pubertal growth spurt) and MP3-I (end of the pubertal growth spurt), which is called the MP3-HI stage has been introduced.

MP3-F stage: Corresponds to the start of the curve of pubertal growth spurt.

Features observed by Hagg and Taranger:

- Epiphysis is as wide as metaphysis.
- Additional features are observed in this method.
- Ends of epiphysis are tapered and rounded.
- Metaphysis shows no undulation.

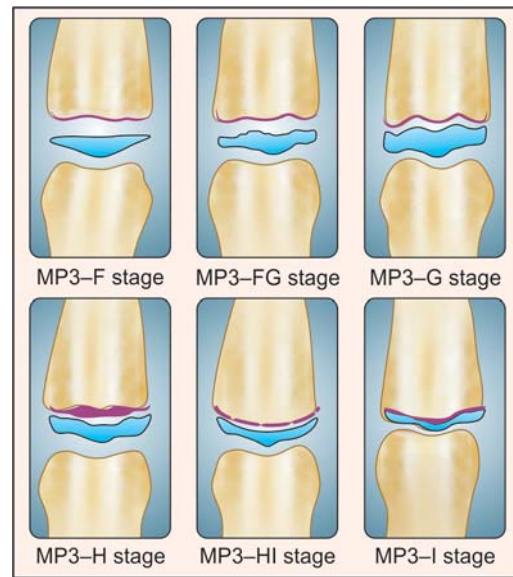


Fig. 10.6: Modified Hagg and Taranger method

- Radiolucent gap (representing cartilaginous epiphyseal growth plate) between epiphysis and metaphysis is wide.

MP3-FG stage: Acceleration of the curve of pubertal growth spurt.

Features observed by Hagg and Taranger:

- Epiphysis is as wide as metaphysis.
- Distinct medial and/or lateral border of epiphysis forms line of demarcation at right angles to distal border.
- Additional features are observed in this method.
- Metaphysis begins to show slight undulation.
- Radiolucent gap between metaphysis and epiphysis is wide.

MP3-G stage: Maximum point of pubertal growth spurt

Features observed by Hagg and Taranger:

- Sides of epiphysis have thickened and cap its metaphysis, forming sharp distal edge on one or both sides.
- Additional features are observed in this method.
- Marked undulations in metaphysis give it a "Cupid's Bow" appearance.
- Radiolucent gap between epiphysis and metaphysis is moderate.

MP3-H stage: Deceleration of the curve of pubertal growth spurt.

Features observed by Hagg and Taranger:

- Fusion of epiphysis and metaphysis begins. Additional features are observed in this method.
- One or both sides of epiphysis form obtuse angle to distal border.
- Epiphysis is beginning to narrow.
- Slight convexity is seen under central part of metaphysis.
- Typical "Cupid's bow" appearance of metaphysis is absent, but slight undulation is distinctly present.
- Radiolucent gap between epiphysis and metaphysis is narrower.

MP3-II stage: Maturation of the curve of pubertal growth spurt.

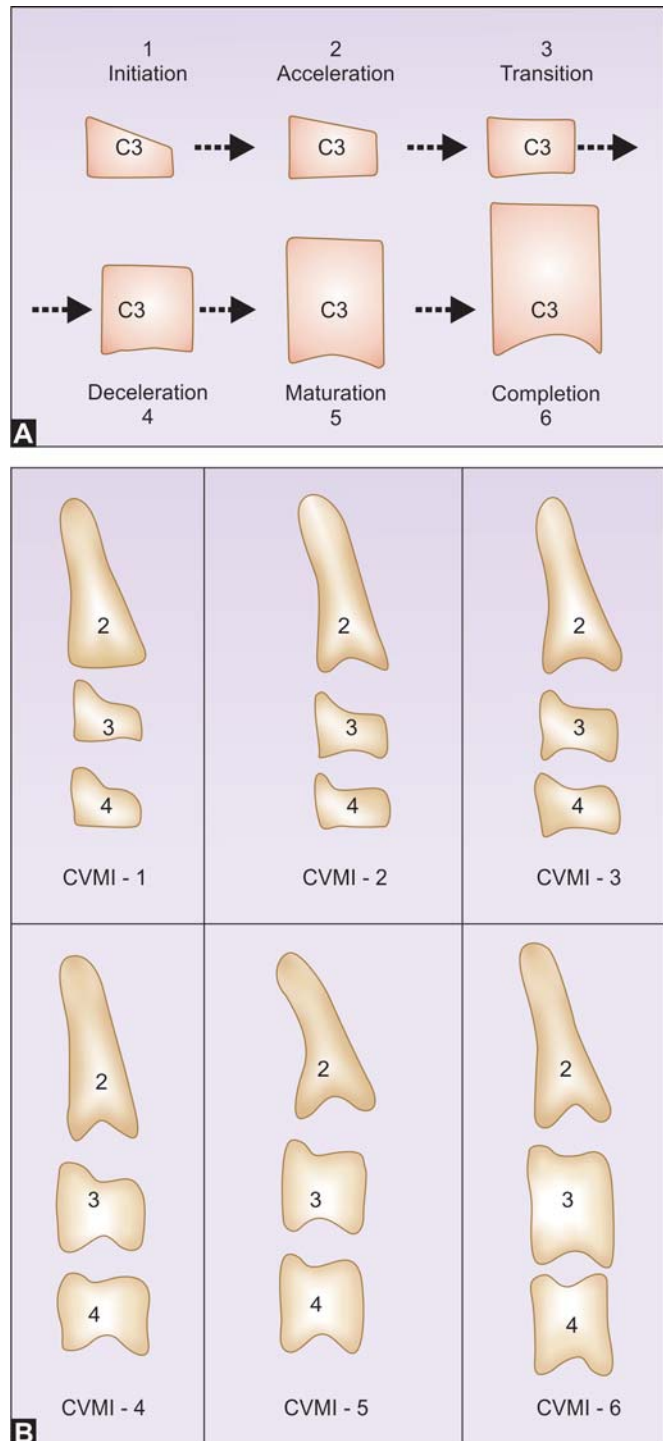
Features of this "new" stage observed in this study:

- Superior surface of epiphysis shows smooth concavity.
- Metaphysis shows smooth, convex surface, almost fitting into reciprocal concavity of epiphysis.
- No undulation is present in metaphysis.
- Radiolucent gap between epiphysis and metaphysis is insignificant.

Cervical Vertebrae as Skeletal Maturity Indicator

Hassel and Farman (1995) found that the shapes of the cervical vertebrae were found to differ with different levels of skeletal development (Figs 10.7A and B).

- *Initiation:* Inferior borders of the second, third and fourth cervical vertebrae are flat at this stage. The third vertebra is wedge shaped and the superior vertebral borders are tapered from posterior to anterior. 100 percent of pubertal growth remains.
- *Acceleration:* Concavities on the inferior borders of second and third vertebrae begin to develop. Inferior borders of the fourth vertebrae remain flat. Vertebral bodies of third and fourth vertebrae are nearly rectangular in shape. 65 to 85 percent of pubertal growth remains.
- *Transition:* Distinct concavities are shown on the inferior borders of second and third vertebrae. A concavity begins to develop on the inferior border of fourth vertebra. Vertebral bodies of third and fourth are rectangular in shape. 25 to 65 percent of growth remains.



Figs 10.7A and B: (A) The stages of cervical vertebrae maturation and (B) Diagrammatic representation of cervical vertebrae maturation

- **Deceleration:** Distinct concavities can be observed on the inferior borders of second, third and fourth cervical vertebrae. Vertebral bodies of third and fourth begin to be more square in shape. 10 to 25 percent of pubertal growth remains.
- **Maturation:** Marked concavities are observed on the inferior border of second, third and fourth cervical vertebrae. Vertebral bodies of third and fourth are almost square in shape. 5 to 10 percent of pubertal growth remains.
- **Completion:** Deep concavities are observed on the second, third, and fourth cervical vertebrae. Vertebral bodies are greater vertically than horizontally. Pubertal growth has been completed.

Modified Stages of Cervical Vertebral Maturation given by McNamara, Bacetti and Franchi (2005)

The six stages are defined as follows (Fig. 10.8):

Cervical stage 1 (CS1): The lower borders of all the three vertebrae (C2-C4) are flat. The bodies of both C3 and C4 are trapezoid in shape (the superior border of the vertebral body is tapered from posterior to anterior). The peak in mandibular growth will occur on an average two years after this stage.

Cervical stage 2 (CS2): A concavity is present at the lower border of C2. The bodies of both C3 and C4 are still trapezoid in shape. The peak in mandibular growth will occur, on an average within one year after this stage. Class III treatment with maxillary expansion and protraction is effective in the maxilla only when it is performed before the peak (CS1 or CS2), whereas it is effective in the mandible during both prepubertal and pubertal stages.

Cervical stage 3 (CS3): Concavities at the lower borders of both C2 and C3 are present. The bodies of C3 and C4 may be either trapezoid or rectangular horizontal in shape. Stage CS3 represents the ideal stage to begin functional jaw orthopedics, as the peak in mandibular growth will occur within the year or after this stage. CS3 is also the appropriate age for treatment of vertical malocclusion, because peak in mandibular growth occurs during this stage.

Cervical stage 4 (CS4): Concavities at the lower borders of C2, C3, and C4 are now present. The bodies of both C3 and C4 are rectangular and horizontal in shape. The peak in mandibular growth has occurred within one or two years before this stage.

Cervical stage 5 (CS5): The concavities at the lower borders of C2, C3, and C4 still are present. At least one of the bodies of C3 and C4 is square in shape. If not square, the body of the other cervical vertebra is still rectangular and horizontal. The peak in mandibular growth ends at least one year before this stage.

Cervical stage 6 (CS6): The concavities at the lower borders of C2, C3, and C4 still are evident. At least one of the bodies of C3 and C4 is rectangular and vertical in shape. If not rectangular and vertical, the body of the other cervical vertebra is square. The peak in mandibular growth end at least two years before this stage.

Mandibular Canine Calcification as an Indicator of Skeletal Maturation

Coulthino, Buschang and Miranda studied the association between the canine calcification and epiphyseal-diaphyseal stages of ossification for third proximal, middle, and distal phalanges and fifth proximal phalanx. The stages were assessed using Hagg and Taranger method. A close association was found between the stages of mandibular canine calcification and the skeletal maturity indicators (Figs 10.9A and B).

- Canine stage F indicates the initiation of puberty.
- The timing of stage G coincides with the capping of the third, middle and the fifth proximal phalanges and the presence of the adductor sesamoid. It is indicative of PHV.
- The intermediate stage between stages F and G should be used to identify the early stages of the pubertal growth spurt.

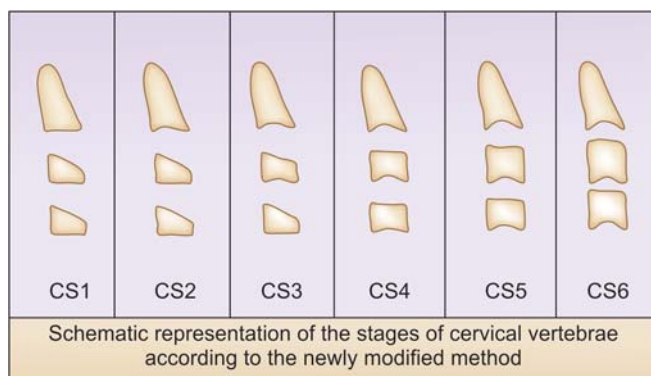


Fig. 10.8: Modified cervical vertebrae maturation stages

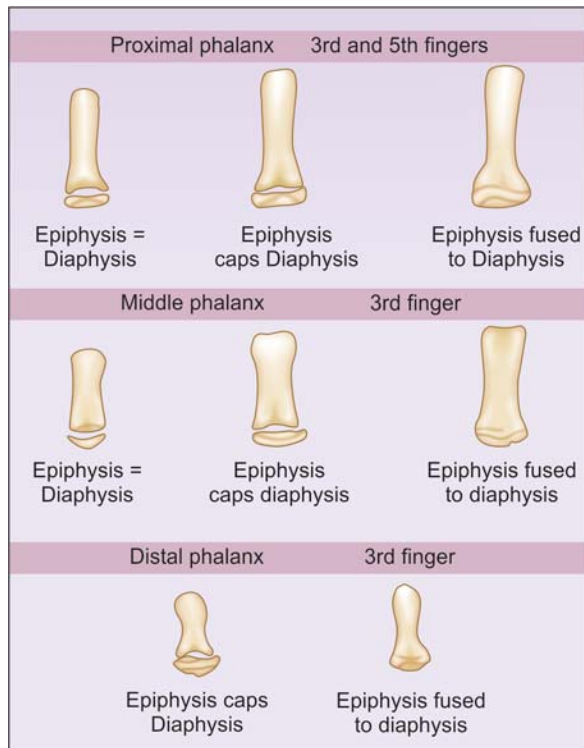


Fig. 10.9A: Epiphyseal-diaphyseal stages of ossification for third proximal, middle, and distal phalanges and fifth proximal phalanx

While the findings clearly indicate a relationship, canine development cannot and should not be used as the sole criterion to predict developmental landmarks (i.e. onset of puberty, PHV). Dental calcification stages of the mandibular canine provide readily available and easily recognized indications of the maturity status of a person; they are simple first-level diagnostic tests to determine whether additional, more sensitive, measures of maturity are warranted.

Development of Mandibular Third Molar as an Indicator of Skeletal Maturation

Engstrom et al in 1983 did a study correlating the developmental stages of mandibular third molar with skeletal age assessed by hand wrist radiographs. The development stages of third molar were categorized into one of the following classes (Figs 10.10A to E):

- A: Tooth germ visible as a rounded radiolucency.
- B: Cusp mineralization complete.

C: Crown formation is complete.

D: Root half formed.

E: Root formation complete, but apex not closed.

The skeletal development in the hand wrist radiograph is classified as follows:

PP2: Proximal phalanx of second finger, the epiphysis is as wide as the diaphysis.

MP3 cap: Middle phalanx of third finger, the epiphysis caps its diaphysis.

DP3 U: Distal phalanx of the third finger, complete epiphyseal union.

RU: Distal epiphysis of radius, complete epiphyseal union.

After comparing the stages of formation of lower third molar with hand wrist radiographs, the following points were concluded. It was observed at the stage PP2, the lower third molar showed complete crown mineralization in majority of the subjects. At DP3 U stage, the lower third molar crown was still incomplete in some subjects, but it had already attained full root length in others. At stage RU, the crown was completed only in one-third of the subjects and rarely had the root developed in one third, and the root had reached full length in the rest. At MP3 cap, the lower third molar crown formation was complete in the majority of the subjects.

Frontal Sinus as Skeletal Maturity Indicator Ruf and Pancherz—1996

In this method, lateral cephalometric radiographs are used for measuring the size of the frontal sinus at yearly intervals. Radiographs were oriented with the nasion-sella line horizontally. The peripheral border of the frontal sinus was traced, and the highest (Sh) and lowest (S1) points of sinus extension relative to the nasion-sella line were marked. Perpendicular to the interconnecting line (Sh-S1), the maximum width of the frontal sinus was assessed (Fig. 10.11). The average yearly growth velocity (millimeters per year) of the frontal sinus was calculated separately for each of the prediction intervals (T1 or T2). The radiographic magnification of 7 percent was not taken into account.

From longitudinal growth data of the subjects, the average yearly body height growth velocity (millimeters

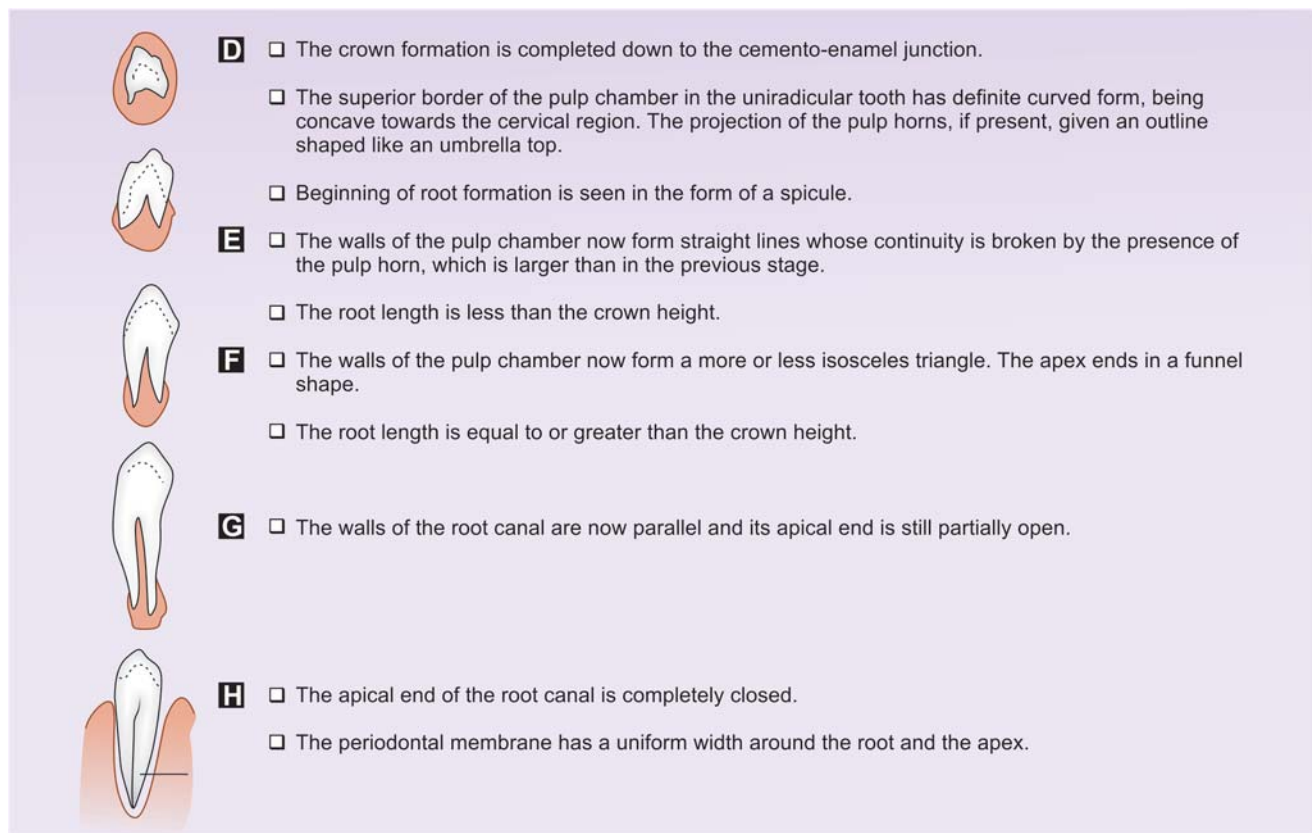
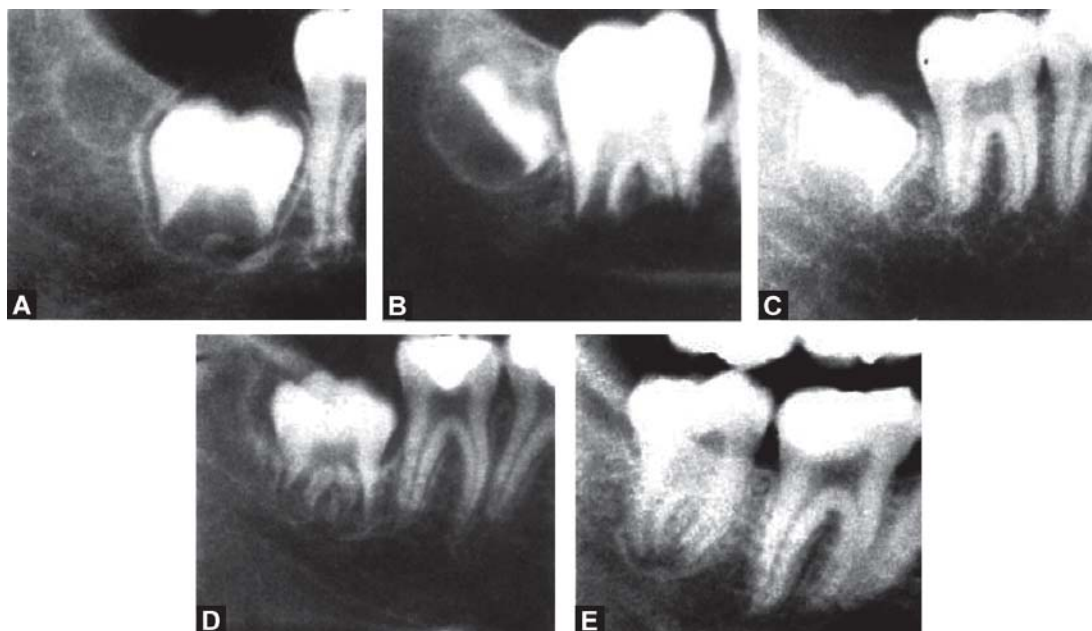


Fig. 10.9B: Stages of canine calcification compared to hand wrist radiograph



Figs 10.10A to E: Developmental stages of the lower third molar: (A) Stage 1: Tooth germ visible as a rounded radiolucency. (B) Stage 2: Cusp mineralization complete. (C) Stage 3: Crown formation complete. (D) Stage 4: Root half formed. (E) Stage 5: Root formation complete, but apex not closed

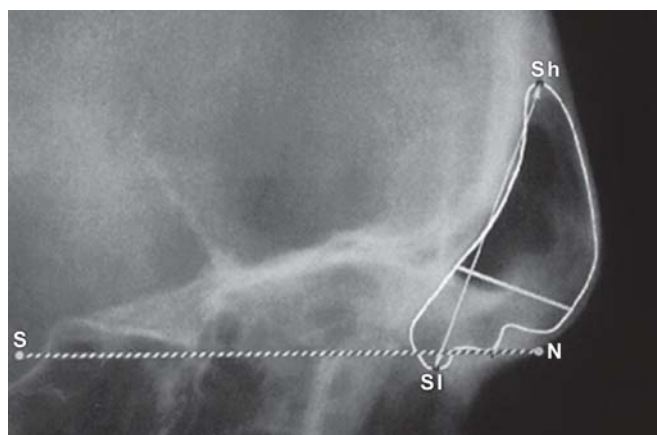


Fig. 10.11: Assessment of maximal frontal sinus width perpendicular to interconnecting line Sh-SI

per year) was calculated. The maximum body growth velocity at puberty was assigned as body height peak (Bp). The body height growth data were used only to test the accuracy of the prediction of pubertal stage as assessed from frontal sinus development.

Ruf and Pacherz compared the relationship between growth of frontal sinus and somatic skeletal maturity and drew the following conclusions.

- Frontal sinus growth velocity at puberty is closely related to body height growth velocity.
- Frontal sinus growth shows a well-defined pubertal peak (Sp), which, on the average, occurs 1.4 years after the pubertal body height peak (Bp).
- In male subjects, the average age at frontal sinus peak is 15.1 years.
- In a one-year observation interval, a peak growth velocity in the frontal sinus of at least 1.3 mm per year is attained by 84 percent of the subjects.
- In a two-year observation interval, a peak growth velocity in the frontal sinus of at least 1.2 mm per year is attained by 70 percent of the subjects.

- These specific frontal sinus growth velocities (1.3 mm/year for the 1-year interval and 1.2 mm/yr. for the 2-year interval) were assigned as threshold values T1 and T2, respectively, for growth prediction.

Prediction Procedure (Table 10.4)

The frontal sinus growth velocity (Sv) in each person of this study was compared with the threshold values T1 (1.3 mm/yr.) and T2 (1.2 mm/yr.). If the frontal sinus growth velocity (Sv) was as high as or higher than the T-value (T1 or T2), it may be expected that frontal sinus peak was reached during the prediction interval. Consequently, it may be assumed that body height peak has been reached approximately 1.4 years before the midpoint of the observation interval. If the frontal sinus growth velocity (Sv) is lower than the T-value (T1 or T2), it is not known whether the subject is prepeak or postpeak in frontal sinus growth. Therefore, the age of the subject is also needed to predict somatic maturity stage. As the frontal sinus growth peak is reached at an average age of 15.1 years, a lower subject age in combination with a Sv lower than the T-value means that the frontal sinus growth peak has not been reached. Consequently, the body height peak has not been reached or has occurred less than 1.4 years before the end of the observation interval (T1 or T2). On the other hand, if the subject's age is higher than 15.1 years, along with a Sv lower than the T-value, then it can be assumed that frontal sinus growth peak had been passed and, consequently, the body height peak had been passed more than 1.4 years before the beginning of the observation interval (T1 or T2).

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Table 10.4: Prediction procedure for somatic maturity based upon the evaluation of frontal sinus growth. The results from the comparison of the frontal sinus growth velocity (Sv) with the respective threshold value (T) and the prediction of body height peak (Bp) are given

Sinus data	Prediction
Sv > T	Bp passed by approximately 1.4 years.
Sv < T and Age < 15.1 years	Bp not yet reached or reached less than 1.4 years before the end of the observation interval.
Sv < T and Age > 15.1 years	Bp passed by more than 1.4 years with respect to the beginning of the observation interval.

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Craniometry, Anthropometry and Cephalometrics in Growth

CHAPTER OUTLINE

- Anthropometry
- Craniometry
 - History of craniometry
 - Craniometric landmarks
 - Craniometric measurements
 - Craniometric indices
- Cephalometrics and Growth
 - Origin of cephalometry
 - Method of cephalometric data acquisition
 - Cephalometric landmarks
 - Reference planes
- Standardizing the Cephalograms
 - Natural head position
 - Norms
 - Superimposition
- Application of Cephalometry in Growth
 - Evaluation of present growth status
 - Growth prediction
 - Identification of areas of growth
 - Identification of timing of growth spurts
 - Soft tissue growth assessment
 - Assessment of late growth changes
 - Differentiating impact of growth from orthodontic treatment

Anthropology is the observation, measurement, and explanation of human variability in time and space. This includes both biological variability and the study of cultural or learned behavior among contemporary human societies. Anthropology has four major subfields: cultural anthropology, physical or biological anthropology, archeology, and linguistics. Physical or biological anthropology, commonly called anthropometrics, literally means man (anthro) measurements (metrics). It is the measurement of the size and proportion of the human

body. Orthodontists are called functioning anthropologists. They measure the bones of the face, skull, and teeth and study the relationships of these structures.

ANTHROPOMETRY

The Frenchman, Alphonse Bertillon, in 1883, gave rise to a system of identification based on the unchanging character of measurement of parts of the human frame. This system came to be called "Bertillonage" and laid the foundation for modern anthropometry. Anthropometrics was first used in the 19th and early 20th century in criminalistics, in identifying criminals by facial characteristics. Anthropometry, a branch of anthropology is the systematized art of measuring and making observations on man, his skeleton, his brain or other organs by the most reliable means and methods and for scientific purposes. This science was further developed by Broca, Campor and Morton.

It includes the following branches:

- a. *Craniometry*: Measurement of skulls.
- b. *Osteometry*: Measurement of skeletal system.
- c. *Cephalometry*: Measurement of head inclusive of soft tissues, be it living or dead.
- d. *Somatometry*: Measurement of living body tissues.

CRANIOMETRY

Craniometry is the study of the shape and form of the human head or skull, sometimes known as craniology. Craniometry as defined by the Edinburgh encyclopedia is the *art of measuring skulls of animals so as to discover their specific differences*. The modern and quantitative study of craniology is derived essentially from the



Fig. 11.1: Apparatus for craniometry devised in 1902

nineteenth century, when it became widely accepted that theories of evolution could be explored through detailed comparisons of skulls (Fig. 11.1).

History of Craniometry

Observations as to variations in human skull form have been recorded for many centuries. Hippocrates (460-357 BC) was a pioneer in the field of anthropology. He had made numerous descriptions about the variety of skull forms. Leonardo da Vinci, in the fifteenth century (1452-1519) was one of the earliest persons to apply the principle of measurement of head. He used a variety of lines related to specific structures in head to assist in the study of human form (Fig. 11.2). Albert Durer (1471-1528) was the first person to write a book on anthropometry. The sixteenth century saw the first scientific attempt at cranial measurement by Spigel (1578-1625). He described four lines: *facial*—from the most inferior point of the chin to the most superior point on the forehead; *occipital*—from the crown of the head to the atlas; *frontal*—from one temple to the other; and *sincipital*—from the lowest part of the ear, in the region of the mastoid process, to the highest part of the sinciput, the sinciput being the anterior part of the head or skull, from the forehead to the crown. Spigel propounded the theory that in a well-proportioned skull, these lines should be equal to one another.

Pieter Camper (1722-1789), a Dutchman introduced the facial angle which was extensively used. The facial

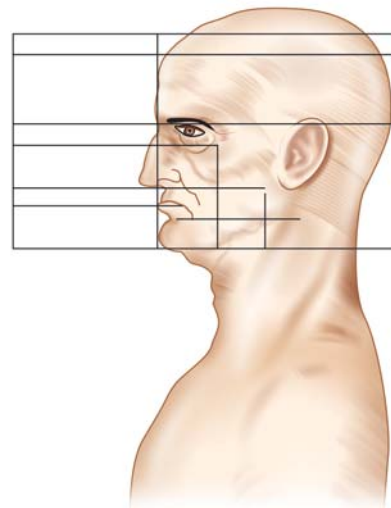


Fig. 11.2: Study of man's head by Leonardo da Vinci

angle was formed by the intersection of facial line and horizontal line. According to Camper, the average facial angle was 70° for African blacks, 80° for Europeans and 58° for orangutans. Deschamps (1740-1824) introduced the cephalic triangle which was made of facial, coronal and occipital angles. Daubenton (1716-1799) and Sir Charles Bell (1744-1842) took special interest in the position of foramen magnum. Johann Friedrich Blumenbach (1752-1840) was a great antagonist to the ideas of Camper. He rejected the methods of lines and angles and completely disagreed with the idea of viewing the skull in *norma lateralis*. He introduced the concept of viewing the skull vertically upon its vault.

Anders Retzius (1796-1860) combined the ideas of Camper and Blumenbach and provided the method which forms the basis for today's methods used in craniology. He also introduced the cephalic index, as the ratio of the breadth of the skull to its length, expressed as percentage. John Barclay (1758-1826) included mandible for the first time into cranial measurements.

The nineteenth century belonged to three eminent men in the field of craniology: Thomas Huxley, Broca, and Topinard. Thomas Huxley (1825-1895) introduced sphenothmoidal and sphenomaxillary angles. Broca founded the Paris Society of Anthropology and was the first person to introduce the craniostat made of wood for positioning and measuring the skulls. He also introduced a new basal line called "plan alveolo-

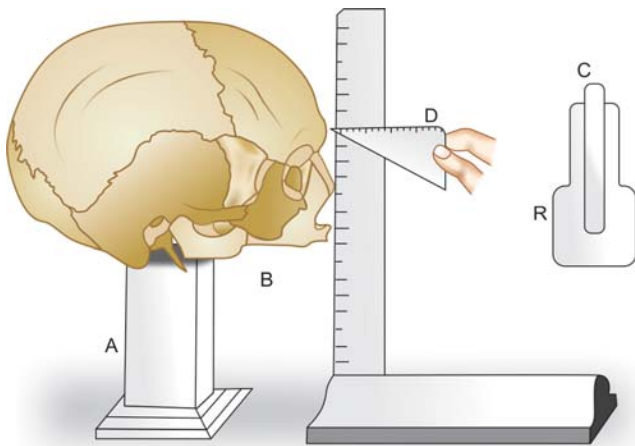


Fig. 11.3: Paul Topinard's craniostat (A—Pedestal; B—Skull placed in shelf; C and R—Portion to support maxilla; D—Graduated rule with a sliding triangle)

condyliens" which passes through the alveolar point and tangential to the inferior surfaces of the two occipital condyles. Paul Topinard (1830-1912) modified the craniostat (Fig. 11.3). He used a pedestal and shelf arrangement, with the skull placed on the shelf. There was a sliding portion which can be moved to support the maxilla. Perpendicular to the shelf is a graduated rule with a sliding triangle used to locate specific points.

The most important event in dentistry took place in Frankfurt-am-Maine in August 1882, at the 13th general congress of the German Anthropological Society. It is to this conference that the Frankfurt Horizontal Plane owes its name. Subsequent to the agreement, the horizontal plane is now taken as passing through right and left porion and left orbitale. With the discovery of X-rays by Professor Wilhelm Conrad Roentgen in 1895 and invention of cephalostat by Holly Broadbent and Hoffrath, radiographic cephalometry became more popular and replaced craniometry in clinical practice to a very great extent.

Craniometric Landmarks (Figs 11.4 and 11.5)

The practice of craniometry consists of taking precise measurements using 'landmarks' on the skull. The areas where these bones meet in the skull can be easily identified, and these places form many of the major landmarks of the skull, for example, the 'bregma', where the two parietals and the frontal meet, which is in effect the highest part of the skull. The distances between the various points can be measured, thus forming the basis

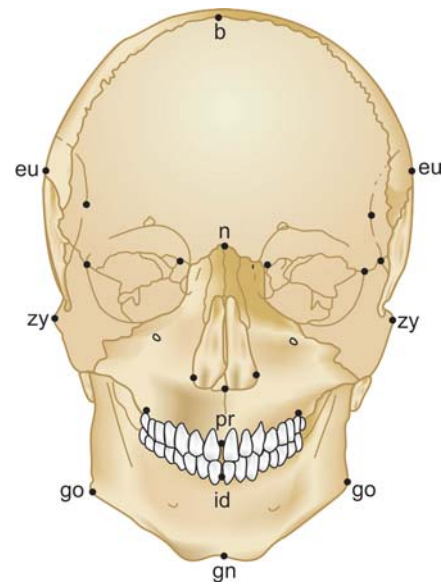


Fig. 11.4: Anatomical landmarks of the skull—anterior view (From Data Collection Procedures for Forensic Skeletal Examination by Peer H Moore-Jansen, Richard L Jantz, Stephen D Ousley, 1994)

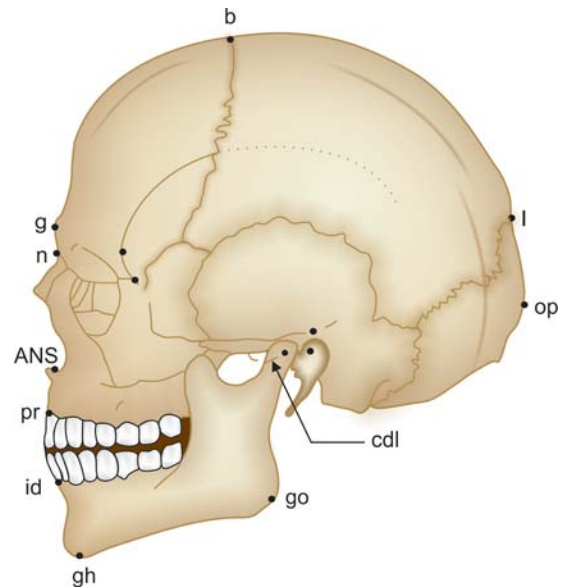


Fig. 11.5: Anatomical landmarks of the skull—lateral view (after Moore-Jansen et al 1994)

of craniometry. In this way, a structural model of the skull, consisting of the angles and lengths between the landmarks, can be formed, and thus making it possible to compare one skull with another, and to make statistical comparisons between populations. Comparisons can also

be made between human populations. People from different parts of the world look different, and this is reflected in their cranial anatomy. For example, modern European skulls are characterized by large faces and noses, and relatively long skulls; modern East Asians are lightly built, with very short, flat faces. Craniometry allows these similarities and differences to be treated not as racial types, but as patterns of biological variation, and to be understood in terms of history and adaptation.

BREGMA (b): The ectocranial midline point where the coronal and sagittal sutures intersect.

EURION (eu): Instrumentally determined ectocranial points on opposite sides of the skull that form the termini of the line of greatest cranial breadth (Paired landmark).

GNATHION (gn): The most inferior midline point on the mandible.

GONION (go): A point along the rounded postero-inferior corner of the mandible between the ramus and the body. To determine the point, imagine extending the posterior ramus border and the inferior corpus border to form an obtuse angle. The line bisecting this angle meets the curved gonial edge at gonion (Paired landmark).

INFRADENTALE (id): The midline point at the superior tip of the septum between the mandibular central incisors.

NASION (n): The point of intersection between the frontonasal suture and the internasal suture at midsagittal plane.

PROSTHION (pr): The most anterior point in the midline on the alveolar processes of the maxillae.

ZYGION (zy): Instrumentally determined as the most lateral point on the zygomatic arch (Paired landmark).

CONDYLION LATERALE (cdl): The most lateral point on the mandibular condyle (Paired).

GLABELLA (g): The most anterior midline point on the frontal bone, usually above the frontonasal suture.

LAMBDA (l): The ectocranial midline point where the sagittal and lambdoidal sutures intersect. If location of this point is rendered difficult by the presence of wormian bones, locate the point where projections of the sagittal and lambdoid sutures would meet.

NASOSPINALE (ns): The point where a line drawn between the inferior most points of the nasal (piriform)

aperture crosses the midsagittal plane. Note that this point is not necessarily located at the tip of the nasal spine.

OPISTHOCCRANION (op): Instrumentally determined most posterior point of the skull on the external occipital protuberance.

BASION (ba): The midline point on the anterior margin of the foramen magnum. For cranial height measurements, the point is placed on the anteroinferior portion of the foramen's rim. For basinasal and basiprostion measurements, the point is located on the most posterior point on the foramen's anterior rim and is sometimes distinguished as endobasion.

ORALE: is the midpoint of a line connecting the posterior borders of the sockets of the upper central incisors. This point can also be located approximately in the living.

STAPHYLION: is the midline point of a line connecting the most anterior points of the posterior border of the hard palate on either side. This point is situated at the base of the variable posterior nasal spine.

Anthropometric Instruments

The basic craniometric measuring instruments used are (Figs 11.6A and B):

- Sliding anthropometric caliper
- Spreading anthropometric caliper
- Anthropometer
- Tape measure
- Skin fold caliper
- Weight scale
- Head spanner.

Anthropometer consists of hollow sliding rods, graduated in millimeters, used for taking various measurements including vertical and transverse body measurements. Spreading calipers are employed to measure head and face diameters. The rounded tip of each caliper horn is held between thumb and forefinger of each hand. Sliding compass is used to measure smaller diameters of the head and head spanner is used to determine height of the head (Fig. 11.6C).

Todd's head spanner, the horseshoe-shaped frame is placed over the skull and the two opposing pins are positioned in the earholes. Then the folding tab on one side of the frame is placed at the base of the left orbit, bringing the instrument into alignment. The vertical ruler can then be used to measure the cranium.

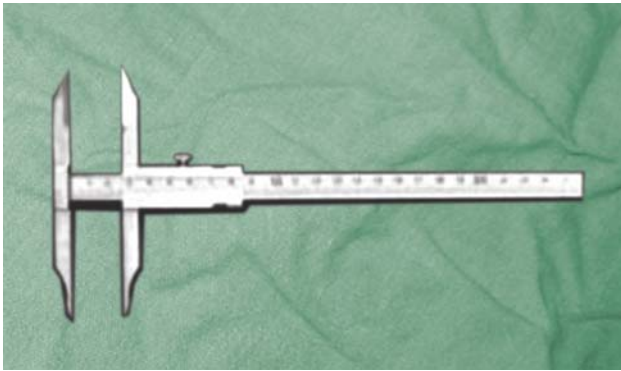


Fig. 11.6A: Sliding caliper



Fig. 11.6B: Spreading caliper



Fig. 11.6C: Head spanner

Facial orthometer: Designed by William A. Elasser, is a simple instrument which facilitates the application of anthropometric methods to population studies.

It is an instrument which measures dentofacial morphology, and the measurements may be used for quantitative classification of malocclusion. The construction and use of this instrument follows accepted anthropometric principles.

The maxillator (Fig. 11.6D): The Salzman Maxillator provides a means of measuring the face and its

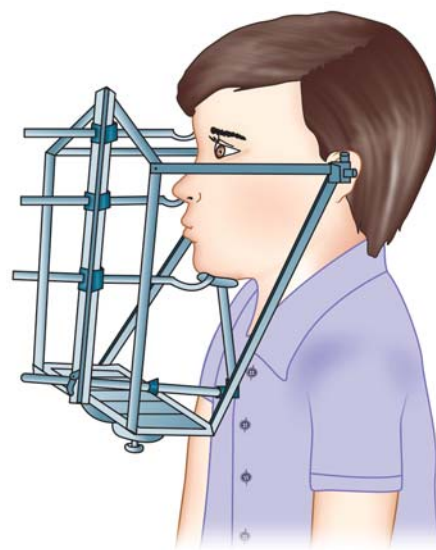
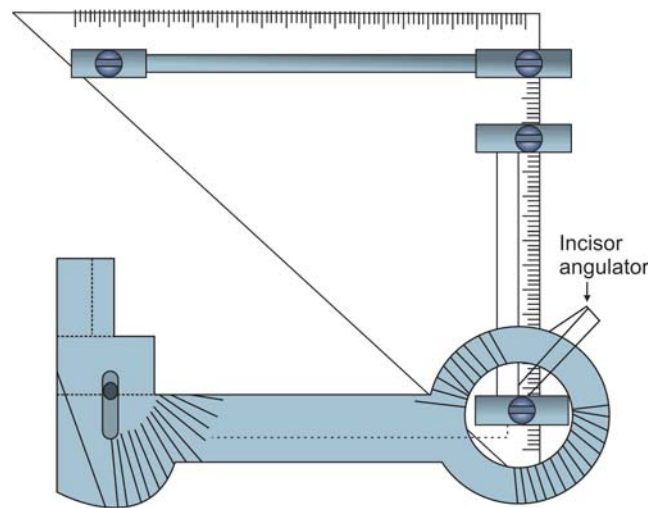


Fig. 11.6D: Maxillator

components, including the jaws, jaw bones, their parts, processes, and anthropologic, orthodontic and other bone and soft tissue landmarks.

It is used for obtaining measurements on the growth and development of the face before beginning treatment, diagnosis, treatment planning and prognosis. It is used for obtaining facial measurements including angles, planes and indices to serve as guides in restoring facial contours by plastic surgeries as in skin and muscle grafting. It is also used in the construction of full or partial fixed or removable dentures and dental restorations, for obtaining data in identification of faces, skulls and their component parts. Frankfort-Mandibular plane angle can be obtained quickly and directly on the patient. Incisor-Mandibular plane angle given by Margolis can also be measured.

Definitions of Cranial and Postcranial Measurements (Figs 11.7 to 11.9)

- *Maximum cranial length (g-op)*: Distance between glabella (g) and opisthocranium (op) in the midsagittal plane, measured in a straight line.
Instrument used: Spreading caliper.
- *Maximum cranial breadth (eu-eu)*: Maximum width of skull perpendicular to midsagittal plane wherever it is located, with the exception of the inferior temporal lines and the area immediately surrounding them.
Instrument used: Spreading caliper.

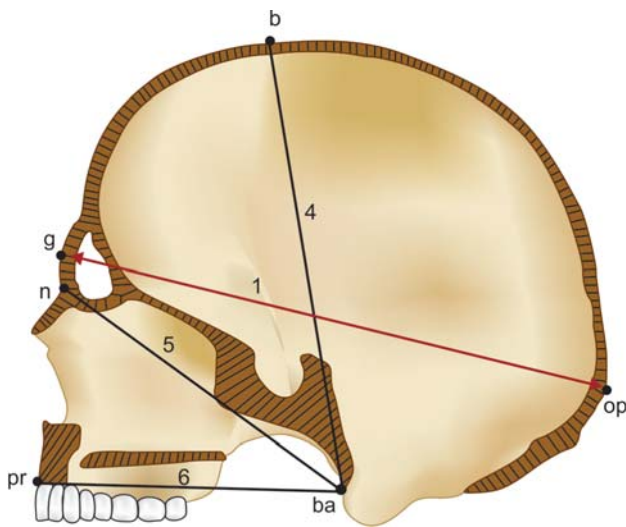


Fig. 11.7: Craniometric measurements in norma lateralis

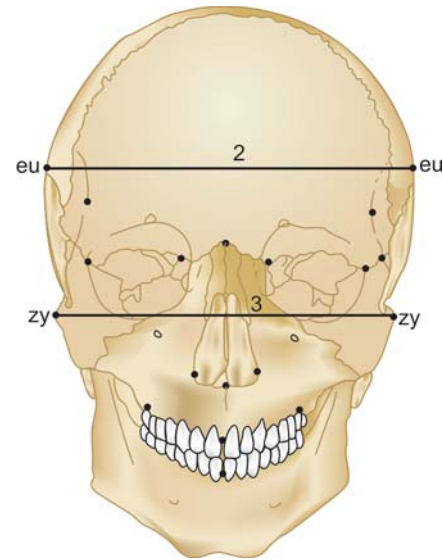


Fig. 11.8: Craniometric measurements in norma frontalis

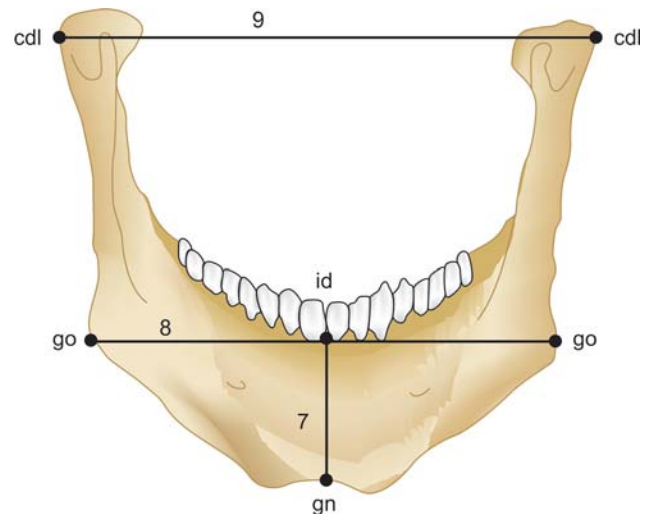


Fig. 11.9: Craniometric measurements in mandible

- *Bizygomatic diameter (zy-zy)*: Direct distance between most lateral points on the zygomatic arches (zy-zy).
Instrument used: Spreading or sliding caliper.
- *Basion-Bregma height (ba-b)*: Direct distance from the lowest point on the anterior margin of foramen magnum (ba), to bregma (b).
Instrument used: Spreading caliper.

Table 11.1: Cranial index and head types

Females	Males	Scientific term	Meaning	Alternative term
< 75%	< 65%	dolichocephalic	long-headed	mesocranial
75–80%	65–75%	mesocephalic	medium-headed	mesaticephalic
> 80%	> 75%	brachycephalic	short-headed	brachycranial

- **Cranial Base length (ba-n):** Direct distance from nasion (n) to basion (ba).
Instrument used: Spreading caliper.
- **Basion-Prosthion length (ba-pr):** Direct distance from basion (ba) to prosthion (pr).
Instrument used: Spreading or sliding caliper.
- **Chin height (id-gn):** Direct distance from infradentale (id) to gnathion (gn).
Instrument used: Sliding caliper.
- **Bigonial width (go-go):** Direct distance between right and left gonion (go).
Instrument used: Sliding caliper.
- **Bicondylar breadth (cdl-cdl):** Direct distance between the lateral most points on the two condyles (cdl).
Instrument used: Sliding caliper.
- **Palate length** is the distance between orale and staphylion.
- **Palatal breadth** is the distance of the inner borders of the sockets from the two upper second molars (endomolaria).
- **Palate height** is the distance of the maximum arching of the palate from the line connecting the two endomolaria.

Craniometric Indices

To describe proportions independent of absolute size, indices are used in anthropology. An index is defined as the ratio of smaller to a larger linear measurement expressed in terms of a percentage. The important indices used are:

- **Cephalic index:** Cephalic index is the ratio of the maximum width of the head to its maximum length, sometimes multiplied by 100 for convenience. Maximum cranial width is measured between eurion to eurion and maximum cranial length is measured between nasion and opisthocranion. It was once widely used to categorize human populations, but is no longer used except for describing individuals' appearances, and has no correlation with behavior. Human populations were characterized as either dolichocephalic (long headed), mesocephalic (moderate headed), or brachycephalic (broad headed). Cephalic indices are grouped as in Table 11.1.
- **Classification of skull according to the height of cranial vault as seen in profile:** This index gives the ratio of a basion-bregma height to maximum cranial length. The formula for computing the index is as follows:

$$\frac{\text{Basion-bregma}}{\text{Maximum cranial length}} \times 100$$

According to this index, the skulls are classified as chamecephalic or low skulls, hypsicephalic or high skulls and a middle type is called as orthocephalic.

 - Chamecephalic X–69.9
 - Orthocephalic 70.0–74.9
 - Hypsicephalic 75.0–X
- **Classification of skull according to the height of cranial vault as seen in anterior view:** This index gives the ratio of basion-bregma height to maximum cranial breadth. The skulls are classified as tapeinocephalic or lowly arched skulls, acrocephalic or high arched skulls and middle class or metriocephalic.
 - Tapeinocephalic X–91.9
 - Metriocephalic 92.0–97.9
 - Acrocephalic 98.0–X
- **Facial index:** The Facial Index is calculated using the formula:

$$\text{Facial Index} = \frac{\text{Nasion-Gnathion Height}}{\text{Bizygomatic breadth}} \times 100$$

Facial index characterizes the proportions of the face. The index shows whether the faces is high and narrow (leptoprosopic), or low and wide (euryprosopic). Mesoprosopic is the middle type.

 - Euryprosopic 80.0–84.9
 - Mesoprosopic 85.0–89.9
 - Leptoprosopic 90.0–94.9
- **Palatine index:** Palatine index is calculated using the formula:

$$\frac{\text{Palate breadth}}{\text{Palate length}} \times 100$$

This index enables the identification of skulls with narrow palate (leptostaphyline) and those with wide palate (brachystaphyline).

- Leptostaphyline X–79.9
- Mesostaphyline 80.0–84.9
- Brachystaphyline 85.0–X
- *Palate height index*: This index enables to find out the arching of palate. It is calculated using the formula:

$$\frac{\text{Palate height}}{\text{Palate breadth}} \times 100$$

A low palate is called chamestaphyline, high palate is hypsistaphyline and intermediate type is called orthostaphyline.

- Chamestaphyline X–27.9
- Orthostaphyline 28.0–39.9
- Hypsistaphyline 40.0–X
- *Total profile angle*: According to this angle, skulls are classified as prognathous if the jaws protrude strongly, orthognathous if the jaws do not protrude, the intermediate type is called mesognathous. Total profile angle is the angle between a line connecting nasion and prosthion and the FH plane.
 - Prognathous 70.0–79.9°
 - Mesognathous 80.0–84.9°
 - Orthognathous 85.0–92.9°
- *Rivet's angle*: This is done to analyze the position of upper face. It is calculated by measuring the angle of the triangle nasion-prosthion-basion at the prosthion.
 - Prognathous X–69.9°
 - Mesognathous 70.0–72.9°
 - Orthognathous 73.0–X

Advantages

- It can be performed on either the living or dried subject specimen.
- It shows the trends of rates, total amount and relative directions of growth in the same person.
- This longitudinal approach is dynamic in which serial measurements can be made of the same growing individual and the actual amount of growth can be evaluated.

Disadvantages

- Sensitivity of some of the tissues to direct measurements like eyes.

- Compressibility of the soft tissues is another source of error inherent to anthropometric technique, i.e. differences in tissue thickness and consistency.
- Amount of pressure exerted on the calipers during measurement further compounds the problem.
- Anthropometry of the dried specimen has the disadvantage of the static cross-sectional type of study.
- In spite of the accuracy of the anthropometric apparatus, exact measurements of growth are extremely difficult to obtain.

CEPHALOMETRICS AND GROWTH

The human skull undergoes active growth from infancy to childhood to adolescence. The growth of the skull is characterized by the descent of maxilla from the cranial base which leads to the secondary displacement of mandible. Simultaneously, the mandible also grows and descends from the maxilla. The downward and forward growth of the maxilla occurs in three dimensions and it is a combination of translation and rotation. Cephalometric radiographs provide relatively valid and practical means of measuring, detailing and interpreting the craniofacial growth. Cephalograms have been extensively used in growth studies even before their role in diagnosis and treatment planning was recognized and promoted. In fact, the original purpose of cephalometrics was only to study growth changes. Cephalometric radiographs were taken at various intervals and they are compared and the differences analyzed by cephalometrists. The cephalometric radiographs have to be superimposed in certain areas which are least affected by growth to evaluate growth occurring elsewhere. Cephalometry permits the individual to be assessed repeatedly at various points of time, i.e. repeated data of the individual can be obtained in longitudinal studies.

Skeletal, dental and soft tissues of the craniofacial complex have been defined by cephalometric methods and the norms established. The norms are age, sex and population specific. By comparing the patient's data to these norms, an objective assessment of the growth is made possible. This section analyses the role of cephalometrics in studying and interpreting growth.

Origin of Cephalometry

X-rays were discovered in 1895 by Roentgen and it enabled the clinicians to visualize the facial skeleton on

a two-dimensional image obtained on the film. The roentgenographic cephalometric technique was introduced to Orthodontics by Holly Broadbent of USA and Herbert Hofrath of Germany in 1931. Broadbent developed a head positioning device called Cephalostat which he used to obtain lateral and anteroposterior views of patient's skull. Cephalometric radiographs have become an integral part of orthodontic practice since then. Cephalometric radiographs enable the clinicians to quantify facial and dental relationships. It gives information about the spatial relationship of superficial and deep structures.

Types of Cephalograms

- **Lateral cephalogram:** The lateral cephalogram is usually taken in closed mouth position. It may also be taken in postural rest position.
- **Frontal or Anteroposterior cephalogram:** In this technique, head is rotated by 90 degrees so that the central ray perpendicularly bisects the transmeatal axis. Frankfurt horizontal plane should be accurately horizontal to get unaltered picture.
- **Oblique cephalogram:** Right and left oblique cephalograms are taken at 45 and 135 degrees to the lateral projection.

Method of Cephalometric Data Acquisition

The basic components for producing a lateral cephalogram are:

- **X-ray apparatus:** It comprises of an X-ray tube, transformers, filters, collimators, and a coolant system all encased in the machine's housing.
- **Image receptor system:** It requires a complex image receptor system that consists of an extra oral film, intensifying screens, cassette, grid, and a soft tissue shield.
- **Cephalostat:** Cephalostat is the head holder. It positions the patients head in three dimensions to receive the X-ray beam. The X-ray source is placed 5 feet or 60 inches away from the patient's mid-sagittal plane. This is done to reduce the magnification. The film is placed 18 cms away from the mid-sagittal plane. Patients' Frankfurt horizontal plane is oriented parallel to the floor by means of ear rods inserted into the external acoustic meatuses and the orbitale pointer (conventional method). Mid-sagittal plane is

parallel to the cassette for the lateral cephalogram. It is perpendicular to the cassette for the postero-anterior cephalogram. The upper part of the face is supported by the forehead clamp positioned at the nasion. The X-ray generator is a step down transformer which generates electric current (10–15 mA, 70–80 kVp) and with medium speed films and intensifying screens, the exposure time is 0.6–1.2 seconds. It is shorter when high speed films are used. Current technical specifications are 80 kVp; 8 mA and 0.8 second exposure time. Some amount of magnification invariably occurs with this technique. Acceptable magnification of the cephalogram is in the range of 5–7 percent. By convention, cephalograms are taken of the left side of the skull. The film size is 8 × 10 inches and the film is placed in the cassette along side the intensifying screen.

Cephalometric Landmarks (Fig. 11.10)

Landmarks on Cranial Skeleton

Ba (Basion)—The lowermost point on the anterior margin of the foramen magnum in the midsagittal plane.

Bo (Bolton point)—The highest point in the upward curvature of the retrocondylar fossa.

G (Bony glabella)—The most anterior point of the frontal bone.

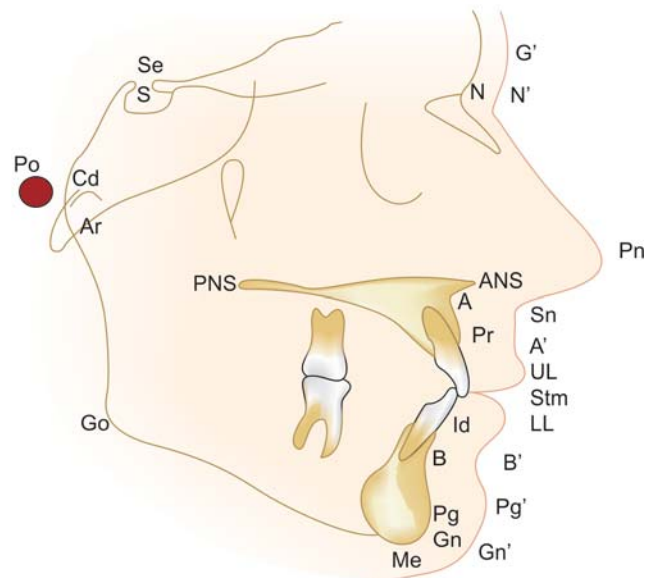


Fig. 11.10: Cephalometric landmarks

Na (Nasion)—The intersection of the internasal suture with the frontonasal suture in the midsagittal plane.

Po (Porion)—Anatomic porion is defined as the superior most point on the upper edge of the external auditory meatus.

Ptm (Pterygomaxillary fissure)—The projected contour of the fissure; the anterior wall represents closely the retromolar tuberosity of the maxilla, and the posterior wall represents the anterior curve of the pterygoid process of the sphenoid bone.

S (Sella)—The midpoint of sella turcica determined by inspection. This point represents the midpoint of the pituitary fossa; it is a constructed point in the median plane.

Se (The point of entrance of the sella)—This point represents the midpoint of the line connecting the posterior clinoid process and anterior opening of the sella turcica; it is at the same level as the jugum sphenoidale and it is independent of the depth of sella.

Landmarks on Maxilla

A-point (Subspinale, ss)—Deepest point on the curvature of the premaxilla between the anterior nasal spine and the crest of the maxillary alveolar process.

ANS (Anterior nasal spine, sp, spinal point)—This point is the tip of the anterior nasal spine seen on the X-ray film from Norma Lateralis.

APMax—Anterior point for determining the length of maxilla. This is constructed by dropping a perpendicular from point-A to the palatal plane (Rakosi).

KR (Key ridge)—The most inferior point on the contour of the shadow of the anterior wall of the infratemporal fossa (Sassouni) in zygomatic buttress.

Or (Orbitale)—The lowest point on the inferior margin of the bony orbit. It is the most inferior point of the orbital rim.

PNS (Posterior nasal spine)—Process formed by the united projecting medial ends of the posterior borders of the two palatine bones. The tip of the posterior spine of the palatine bone in the hard palate.

Pr (Prosthion, superior prosthion or supradentale)—The lowest and the most anterior point on the alveolar portion

of the premaxilla, in the median plane, between the upper central incisors.

Landmarks on Mandible

Ar (Articulare)—The intersection of the lateral radiographic image of the posterior border of the ramus with the occipital bone.

B point (Supramentale, Down's B pt)—The most posterior point in the concavity between infradentale and Pogonion (Downs) or the greatest point of concavity of the mandible between infradentale and Pogonion. It is also defined as the most posterior point in the concavity between the crest of the mandibular alveolar process and pogonion.

Go (Gonion)—The point which on the jaw angle is the most inferiorly, posteriorly, and outwardly directed. It is also defined as the point located by bisecting the angle formed by tangents to the posterior border of the ramus and inferior border of the mandible.

Me (Menton)—The lowermost point on the symphyseal shadow as seen in the Norma lateralis or the most inferior point on the mandibular symphysis in the midsagittal plane.

No (Antegonial notch)—The highest point on the antegonial notch on the lower border of the mandible.

Pg, Pog, or P (Pogonion)—Defined in three ways. The most anterior point of the symphysis or most anterior point in the contour of the chin or most anterior point on the mental protuberance.

Gn, GN or Pgn (Gnathion or Prognathion)—Defined in four ways. The point formed by the intersection of the Y-axis with the bony chin; Most downward and forward point on the symphysis crossing of Y-axis; the most inferior point in the contour of the chin; Midpoint between pogonion and menton at the curvature of the symphysis.

Cd, Co (Condylion)—The superior most point on the outline of the mandibular condyle.

Id (Infradentale)—The highest and the most anterior point in the alveolar process in the median plane between the mandibular central incisors.

Soft Tissue Landmarks

G' (*Glabella*): The most prominent point in the midsagittal plane of the forehead.

N' (*Soft-tissue nasion*): The most concave point in the tissue overlying the area of the frontonasal suture.

Pn (*Pronasale*): The most prominent or anterior point of the nose.

Sn (*Subnasale*): The point at which the columella (nasal septum) merges with the upper lip in the midsagittal plane.

A' (*Soft-tissue A point*): The point of greatest concavity in the midline of the maxillary lip between subnasale and labrale superius.

UL (*Labrale superius*): The anterior most point on the maxillary lip.

Stm (*Stomion*): The inferior most point of the upper lip.

LL (*Labrale inferius*): The anterior most point on the mandibular lip.

B' (*Soft-tissue B point*): The point of greatest concavity in the midline of the lower lip between labrale inferius and soft-tissue pogonion.

Pg' (*Soft-tissue pogonion*): The anterior most point on the soft-tissue chin.

Gn' (*Soft-tissue gnathion*): The point at which the soft-tissue chin intersects the Y-axis.

Reference Planes

The base reference planes provide the observer with a symbolic place to stand upon and make necessary measurements. They serve as the basis for superimposition of serial tracing in an effort to study long-term growth. These base reference lines are used to orient 'before' and 'after' tracings, which analyze therapeutic changes. A reference plane should be as stable as possible and allow interpretations made from its use to coincide with what is actually happening. A reference line for superimposition requires demanding biological support as that landmark must be stable longitudinally, i.e. relatively stable, for there are no absolutely stable lines or points in a biologically dynamic growing person.

Based on orientation and location, they are classified into:

- Horizontal planes
- Vertical planes
- Soft tissue planes.

Sella-Nasion Plane

Commonly used plane for superimposition as growth gets over in the cranial base first. This plane is formed by connecting sella (the midpoint of sella turcica) and nasion.

Frankfurt Horizontal Plane

This is one of the oldest and most widely used plane of cephalometrics. It may be visualized on the living individual, the dried skull, and the lateral cephalogram. It has recently reemerged as a favorite of modern cephalometricians as the best all-around compromise for a base reference line against which other points and lines may be measured for interpretation.

Palatal Plane

This is the line running from the ANS to the PNS and denotes the general limitation of the hard palate superiorly. Palatal plane could be used as the plane of reference for assessing sagittal jaw relationships. The advantages of using palatal plane are:

- Growth changes of point N do not influence the result;
- Rotation of the jaws does not influence the result;
- Inclination of the occlusal plane by dental effects is excluded.

Mandibular Plane

In spite of the fact that mandible is easily seen in the cephalogram, it is surprising that there are four different mandibular plane to describe it. Each one is popular for its own reasons with clinicians. Tweed and Ricketts define the mandibular plane as a straight-line tangent to the lowermost border of the mandible. Downs' one of the founding fathers of clinical cephalometric analysis, defines this plane as the line joining the gonion and menton (Go-Me). A third definition, used by Steiner, is the line joining the gonion and gnathion (Go-Gn). A fourth is Bimler's line joining the menton and the antegonial notch.

STANDARDIZING THE CEPHALOGRAMS FOR COMPARISON

Natural Head Position

Natural head position (NHP) is defined as a standardized and reproducible orientation of head in space when one is focusing at a distant point at the eye level. This is a readily attainable and reproducible position by the subjects when they are positioned in the cephalostat for exposure to the X-ray beam. The search for a reference plane for orienting the crania dates back to 19th century when the craniologists wanted to orient the crania in a standardized manner to facilitate meaningful comparisons. They zeroed up on the Frankfurt horizontal plane passing through porion and orbitale for orienting the crania. Even though it was widely used in craniometry ever since, the Frankfurt horizontal plane presented problems when it was applied while obtaining cephalograms from living subjects. It was assumed that the head is normally positioned when the Frankfurt horizontal plane was parallel to the floor. This was not the case. Neither porion nor orbitale were stable landmarks and some subjects had to tilt their head downward or upwards to make their Frankfurt horizontal plane parallel to the floor. Such cephalograms were of no diagnostic value and the natural head position was hence introduced. Positioning the patients head in natural head position is much more important while obtaining PA cephalograms from the patient. PA cephalograms are mainly taken to register facial asymmetry, and minute change in the head position will affect the radiographic image. Also the ear rods may orient the head inappropriately in the transverse plane due to an abnormal transmeatal axis.

Head positioning for PA cephalogram is identical to that of lateral cephalogram, except that the patient is rotated 90 degrees, i.e. facing the film. To minimize the variation in magnification from patient to patient and to obtain consistent measurement on the same patient over time, many orthodontists choose to keep the distance constant. A distance of 15 cm from the midsagittal plane of the cephalostat to the film cassette is often used.

Norm and Individual Norm

Norms can either be generalized norms for a population or sex specific norms due to sexual dimorphism in

growth. It can also be an age specific norm in growing individuals or a size specific norm due to the variation in the final body size between people.

Norms can be further classified as:

- *Arithmetic Norms:* They are average figures based on angular, linear or proportional measurements. Arithmetic norms provide a valid reference point to compare the subject values. Evaluating cephalogram by angular, linear or proportional measurements have given rise to numerous cephalometric analyses in the literature. The norms were established for the angular and linear measurements in different ways. Some used a population sample of white subjects (Down's) where as others (McNamara) used data from growth studies.
- *Geometric Norms:* Pioneers in cephalometry wanted to compare the subjects cephalograms to clear plastic templates inscribed with standardized facial outlines. Clinicians did not wait for the age, sex and population specific plastic templates to be derived from longitudinal growth studies. They started taking more objective angular and linear measurements from the cephalogram. Geometric norms are average tracings on a transparent sheet of paper which are called the template. They provide a rapid visual orientation of the patient's facial skeleton in relation to the template.

If the patient has a sizable anatomic variation, the cephalometric readings obtained from the patients head film might not correspond to the norm values. Variation in the location of anatomic landmarks such as sella, nasion, porion, orbitale, etc. will result in incorrect interpretations from majority of the cephalometric analysis. So, due care must be exercised in understanding the variations and their geometric consequences in the nature of the cephalometric analyses. In other words, the norms must be modified for individual subject undergoing evaluation of growth (concept of individual norm).

Superimposing Cephalograms for Growth Studies

The face is divided into four zones, namely brain case, cranial base, nasomaxillary complex and the mandible for convenience in the growth studies. The cranial base and the brain case cease to grow during the childhood itself. Nasomaxillary complex grows till the early teens while the mandible might grow till late teens. Mandibular

growth may sometime extend into the third decade of life. Hence, to assess the growth of the face, the cephalometrists zeroed up on the cranial base which serves as a valid reference base since growth ceases early there. The cranial bases also have some midline landmarks visible clearly in the cephalogram which makes angular and linear measurements easier.

Cranial Base Superimposition

The superimposition of two cephalograms taken at two different points of time requires a registration point and a line or plane for superimposition. Most common method of cranial base superimposition is to superimpose along the sella-nasion plane with sella as the registration point. The various methods are:

- Superimposition on the best fit of the anterior cranial base anatomy.
- Superimposition on sella-nasion (SN).
- Superimposition at registration point R with Bolton-nasion planes parallel.
- Superimposition on Basion-nasion plane.

However, tendency of the nasion to undergo resorptive remodeling with time and the broad variability in the length of anterior cranial base (sella-nasion) makes cranial base superimposition error-prone according to some cephalometrists.

Maxillary Superimposition

Maxilla undergoes extensive remodeling during growth. Hence, it is important to assess maxillary growth changes superimposing on maxillary structures. Many methods of obtaining maxillary superimpositions have been suggested. They are:

- Superimposing along palatal plane with ANS as the registration point. It is the most commonly used method and is also called "best fit method".
- Superimposing along the nasal floor with anterior surface of the maxilla as the registration point.
- Superimposing radiographs along the superior and inferior outlines of hard palate.
- Superimposing along the palatal plane similar to the "best fit method" with the registration point on the pterygomaxillary fissure.
- Superimposing along the palatal plane with the registration point at the intersection of outline of infratemporal fossa and hard palate.

Mandibular Superimposition

Mandible is traditionally superimposed along the lower border. Inaccuracy in this method stems from the fact that the gonion undergoes variable backward and vertical displacement during growth. Also, different cephalometrists have defined the mandibular plane through the lower border of the mandible differently:

- Superimposing along the outline of the mandibular canal as this area remodels less than the others as proven by implant studies.
- Superimposing along the lingual cortical contour of the mandible and inferior alveolar canal.
- Superimposing along the lower border of mandible and the inner table of the symphysis.

APPLICATION OF CEPHALOMETRICS IN GROWTH AND DEVELOPMENT

To Evaluate the Present Growth Status of the Individual (To Determine the Skeletal Structure and Facial Type)

Angular Measurements

There is more emphasis on the angular measurements rather than linear measurements in cephalometrics. The face varies very greatly in size between people and it is difficult to establish specific norms for the linear measurements. Angular variables are not subject to the magnification in the cephalogram too. Furthermore, the growing face changes in proportion also as it increases in size. The effects of changing proportions on the cephalometric analysis can be minimized by the careful selection of the angles used.

Saddle angle: This angle is formed by joining sella, nasion and articulare (N-S-Ar). This angle gives an assessment of the relationship between anterior and posterolateral cranial bases. The normal value is $123^{\circ} \pm 5^{\circ}$. The saddle angle is large in retrognathic cases and is small in prognathic cases. Thus, a large saddle angle signifies posterior condylar position and a mandible that is posteriorly placed in relation to cranial base and maxilla.

Articular angle: This angle is formed by joining Sella-Articulare-Gonion (S-Ar-Go). The size of this angle depends on the mandibular position. The angle is large when mandible is retrognathic and small when the mandible is prognathic. The normal value is $143^{\circ} \pm 6^{\circ}$.

Table 11.2: Articular angle alterations and their interpretations

Decrease in articular angle	Increase in articular angle
• Anterior positioning of mandible • Closing the bite • Mesial migration of posterior teeth	• Posterior location of mandible • Opening the bite • Distal driving of the posterior teeth

This angle can be greatly influenced by orthodontic treatment. The effects of changes in articular angle are given in Table 11.2.

Gonial angle: This angle is formed by joining Articulare, Gonion and Menton (Ar-Go-Me). This angle gives information on the form of the mandible and on its growth direction. If this angle is acute or small, especially in the lower component, the direction of mandibular growth is horizontal. This is a favorable condition for anterior positioning of the mandible using activator. In case the angle is large, functional appliances are not to be used for treatment. The normal value is $128^{\circ} \pm 7^{\circ}$.

The other angular measurements (Fig. 11.11) used in evaluate growth status are:

- SNA
- SNB
- Gonial angle (CdGo to GoMe)
- Mandibular plane angle (SN to GoMe)
- Ramus inclination (ramus tangent to SN).

Angular Measurements for Horizontal and Vertical Measurement of Jaw Bases

- **SNA:** The SNA angle expresses the sagittal relationship of the anterior limit of the maxillary apical base as related to the anterior cranial base. It is a large angle in prognathic maxilla and small in retrognathic maxilla. Mean value is 82° .
- **SNB:** The SNB angle expresses the sagittal relationship between the anterior extent of the mandibular apical base and the anterior cranial base. It is large in prognathic and small in retrognathic mandible. Mean value is 80° .
- **Basal Plane Angle: (PAL-MP):** This is the angle between the mandibular plane and the palatal plane. The basal plane angle is small in horizontal growth pattern and large in vertical growth pattern. Mean value is 25° .
- **Inclination Angle:** Drop a perpendicular from N-S line passing through N. This is called the P-N line.

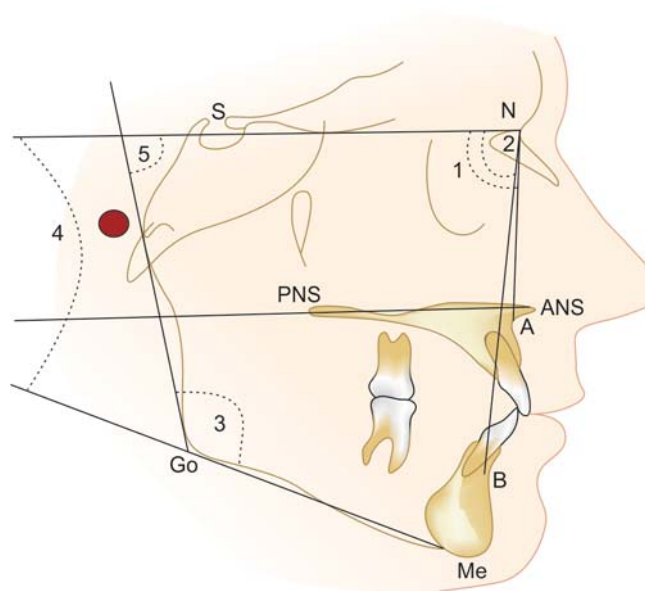


Fig. 11.11: Angular measurements from lateral cephalogram

Now draw the palatal plane. The angle formed between the P-N line and palatal plane is called the inclination of maxillary base. Mean value is 85° .

Linear Measurements (Fig. 11.12)

Linear measurements provide more objective measurement of the growth change in the patient. They are used to quantify the change and the change in actual dimensions. But, they may be affected by magnification and variation in head size between patients.

Cranial base linear measurements:

- **Anterior Cranial Base Length:** This is a measurement between the center of sella to N point. This is useful in comparing the length of jaw bases. Mean value is 71 mm.
- **Posterior (Lateral) Cranial Base Length:** This is the measurement between the sella to articulare. A short posterior cranial base is seen in a vertical growth

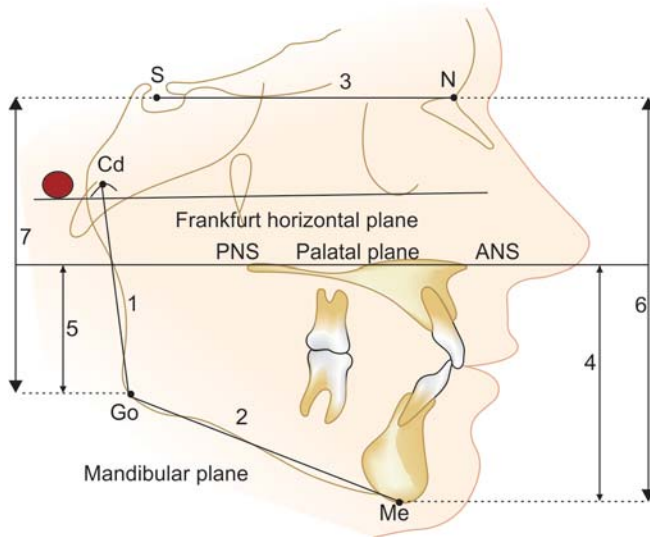


Fig. 11.12: Linear measurements from lateral cephalogram:

1. Cd (Condylion)-Go (Gonion)—Ramus Height
2. Go (Gonion)-Me (Menton)—Corpus length
3. S (Sella)-N (Nasion)—Cranial Base Length
4. LAFH—Lower anterior facial height
5. LPFH—Lower posterior facial height
6. TAFH—Total anterior facial height
7. TPFH—Total posterior facial height

pattern or a skeletal open bite and would give a poor prognosis for functional appliance therapy. Mean value is 32 to 35 mm.

Linear measurement of jaw bases:

- **Extent of Mandibular Base:** This is determined by measuring the distance from gonion to pogonion projected perpendicular to mandibular plane.
- **Extent of the Maxillary Base:** This is determined by measuring the distance between the PNS and point A projected perpendicular on to the palatal plane.
- **Length of Ascending Ramus:** This is the measurement from gonion to condylion. The location of condylion is done by drawing FH plane and intersecting it by a tangent to ramus. The point of intersection represents a constructed condylion.

Facial height: The S-Go length gives us the posterior facial height while the N-Me length gives us the anterior facial height. In a horizontally growing individual, the anterior facial height is greater than the posterior facial height. While in a vertically growing individual, the posterior facial height is greater than the anterior facial

height. In order to estimate the direction of growth we have a ratio called the Jarabak ratio.

$$\frac{\text{Posterior face height}}{\text{Anterior face height}} \times 100$$

A ratio of less than 62 percent indicates a vertical growth pattern while a ratio of more than 65 percent indicates a horizontal growth pattern.

Growth Prediction

Assessment and prediction of dentofacial growth is an important aspect of clinical orthodontics. Although the percentage of adult patients has increased in recent years, the majority of treatment is still directed toward pre-adolescent and adolescent patients. These individuals are undergoing significant growth changes in their occlusions, facial skeletons, and profiles. Such changes are complex because each person has a unique growth pattern influenced by their genetic make-up as well as external environmental factors such as function, disease, habits, and orthodontic treatment.

Cephalometric superimpositions often demonstrate dramatic dental, skeletal, and soft tissue changes during orthodontic treatment. For more detailed description of growth prediction, please refer Chapter on growth prediction.

Identification of the Important Areas of Facial Growth and Quantification of the Growth

- Anterior cranial base—length SN increases by 0.75 mm annually.
- Posterior cranial base—length of posterior cranial base S-Ar increases by 8 mm from age 6 to 16.
- Length of mandibular base—as measured by Go-Pog increases 2 mm for boys and 1.4 mm for girls from age 8 to 16 years.
- Effective length of mandibular base increases by 2 to 3 mm per year.
- Length of maxillary base—as measured by projection of point A to the palatal plane increases by 1.2 mm for boys and 0.8 mm for girls from age 8 to 16 years.
- Effective length of maxillary base increases by 1-2 mm per year.
- Ascending ramus length as measured by Go-Cd increases annually by 2 mm for boys and 1.2 mm for girls.

Table 11.3: Linear and angular variable as per Iowa study

Parameters		Total change	Percentage change		
Age range (years)		5-25	5-10	10-15	15-25
Standing height	Males	59.6 cm	41%	44%	15%
	Females	55.5 cm	54%	41%	5%
ANS-PNS	Males	10.6 mm	37%	42%	21%
	Females	7.2 mm	49%	33%	18%
SNA	Males	1.8%	11%	78%	11%
	Females	0.4%	-25%	50%	75%
Ar-Pog	Males	31.0 mm	34%	39%	27%
	Females	21.0 mm	48%	41%	11%
SN-Pog	Males	5.0°	37%	33%	30%
	Females	3.0°	41%	50%	9%
ANB	Males	-1.6°	-31%	-6%	-63%
	Females	-1.4°	-57%	-71%	28%

Parameters		Total change	Percentage change		
Age range (years)		5-25	5-10	10-15	15-25
Anterior face height (N-Me)	Males	29.8 mm	42%	39%	19%
	Females	21.9 mm	48%	37%	15%
Posterior face height (S-Go)	Males	29.1 mm	35%	36%	28%
	Females	18.5 mm	45%	40%	15%
Soft tissue convexity with nose (GI'-Pr-Prog')	Males	-7.3°	-44%	-70%	14%
	Females	-9.2°	-53%	-37%	-10%
Soft tissue convexity without nose (GI' SLs-Pog')	Males	3.3°	-48%	-36%	185%
	Females	1.0°	-280%	210%	170%

- Mandibular plane angle—angle formed by mandibular plane to palatal plane decreases by 1 degree from mean of 87 degrees (at 9 years) annually.
- Distance from pogonion to nasion perpendicular decreases by 0.5 mm per year.

The Iowa growth study has estimated the percentage change in some linear and angular variables between the age of 5, 10, 15 and 25. Refer Table 11.3.

To Estimate the Timing of Growth Spurts

Structures easily visible in the cephalogram are used for predicting pubertal growth spurt (Fig. 11.13).

Cervical vertebrae: Several clinical studies have shown that the greatest response to functional jaw orthopedics tends to occur during the circumpubertal growth period. Cervical Vertebral Maturation (CVM) method has proved

to be effective to assess the adolescent growth peak, both in body height and mandibular size. Lamparski created separate standards of cervical vertebral maturation for female and male subjects as related to both chronological age and skeletal maturation as observed in the hand-wrist radiograph. The method analyzed size and shape changes in the bodies of five cervical vertebrae (from the second one through the sixth). Greatest increment in mandibular and craniofacial growth occurs during the interval from vertebral stage 3 to vertebral stage 4 (Cvs3 to Cvs4). Baccetti and McNamara have outlined the modified six stages of cervical vertebrae maturation as follows:

Cervical stage 1 (Cvs1): The lower borders of all the three vertebrae (C2-C4) are flat. The bodies of both C3 and C4 are trapezoid in shape (the superior border of the vertebral body is tapered from posterior

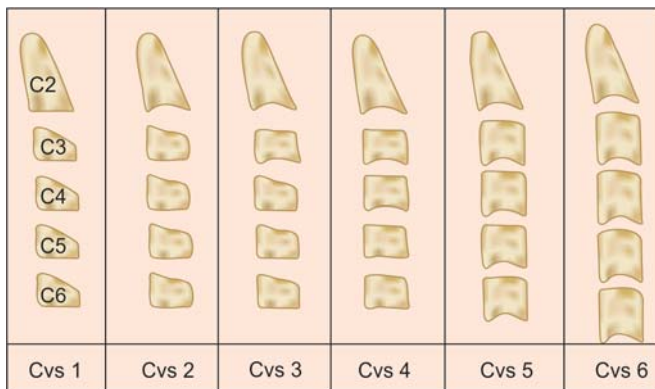


Fig. 11.13: Diagrammatic representation of stages of cervical vertebrae maturation

to anterior). The peak in mandibular growth will occur on average two years after this stage.

Cervical stage 2 (Cvs2): A concavity is present at the lower border of C2 (in four of five cases, with the remaining subjects still showing a cervical stage 1). The bodies of both C3 and C4 are still trapezoid in shape. The peak in mandibular growth will occur on average one year after this stage.

Cervical stage 3 (Cvs3): Concavities at the lower borders of both C2 and C3 are present. The bodies of C3 and C4 may be either trapezoid or rectangular horizontal in shape. The peak in mandibular growth will occur during the year after this stage.

Cervical stage 4 (Cvs4): Concavities at the lower borders of C2, C3, and C4 are now present. The bodies of both C3 and C4 are rectangular horizontal in shape. The peak in mandibular growth has occurred within 1 or 2 years before this stage.

Cervical stage 5 (Cvs5): The concavities at the lower borders of C2, C3, and C4 still are present. At least one of the bodies of C3 and C4 is square in shape. If not squared, the body of the other cervical vertebra still is horizontal and rectangular. The peak in mandibular growth had ended at least 1 year before this stage.

Cervical stage 6 (Cvs6): The concavities at the lower borders of C2, C3, and C4 are still evident. At least one of the bodies of C3 and C4 is vertical and rectangular in shape. If not rectangular vertical, the

body of the other cervical vertebra is squared. The peak in mandibular growth has ended at least two years before this stage.

The depth of the mandibular antegonial notch: Deep notch cases have more retrusive mandibles with a shorter corpus, smaller ramus height, and a greater gonial angle than shallow notch cases. The deep notch sample have less mandibular growth potential as evidenced by a smaller increase in total mandibular length, corpus length, and less displacement of the chin in a horizontal direction than the shallow notch sample. The clinical presence of a deep mandibular antegonial notch is indicative of a diminished mandibular growth potential and a vertically directed mandibular growth pattern.

To Assess the Growth of Soft Tissues

Analysis of the soft tissue should be taken into consideration for the proper evaluation of growth because of individual differences in soft tissue thickness enveloping the craniofacial skeleton. Analysis of soft tissues is more challenging because soft tissues are not as visible as hard tissue structures in the cephalogram. Careful superimposition of the radiographs obtained from longitudinal growth studies has provided the following results. The following conclusion was reached by serial superimposition of cephalograms between 6 to 12 years:

- Length of upper lip grows slightly in length between 6 to 12 years. It is between 1.9 mm to 0.9 mm per year.
- Lower lip growth increments are more from 6 to 12 years. It is between 1.9 mm to 1.5 mm per year.
- The thickness of upper lip increases by 1.1 to 1.4 mm per year. Increase in thickness is only minimal for the lower lip (0.8 mm to 1.2 mm per year)

The data in Table 11.4 was derived from the serial superimposition of cephalograms from age 7 to 18.

To Assess the Change in Dental and Skeletal Relationships during Adult Growth

Rolf Gordon Beherants studied the growth in adult individuals by serial cephalograms and concluded that considerable craniofacial alteration occurs beyond 17 years of age in humans. This is in tune with craniometric observations by Hrdlicka who postulated that growth

Table 11.4: Soft tissue changes following growth

Attainment of adult size for the various soft tissue variables and three skeletal base measurements

	% growth completed at 7 years		Growth ending at or before 15 years		Growth completed past 15 and before 18 years		Growth continuing at 18 years	
	Male	Female	Male	Female	Male	Female	Male	Female
Upper nose height	80	82			×	×		
Lower nose height	67	90		×			×	
Nose depth	63	70		×			×	
Nose skeletal base at pm'-PMV	85	90			×	×		
Upper nose inclination	87	90		×			×	
Lower nose inclination	92	89		×	×			
Upper lip length	88	95	×	×				
Lower lip length	78	91				×	×	
Upper lip thickness at A	73	76		×			×	
Upper lip thickness at LS	82	93		×			×	
Lower lip thickness at LI	85	89		×	×			
Lower lip thickness at B	80	85		×	×			
Sagittal length of mandible at B	75	84		×			×	
Chin thickness at Pgs	80	83			×	×		
Chin thickness at Pg	83	88				×	×	
Symphyseal thickness	92	95		×	×			
Skeletal base at Pg'-PMV	66	74		×			×	
Inclination of skeletal chin	44	27		×	×			
Inclination of chin integument	72	66					×	×

does not cease after attaining adulthood. Beherant's findings can be summarized as follows:

Cranial base: Anterior movement of nasion and a posterior movement of basion and Bolton point occurs at the cranial base. Anterior cranial base from sella to nasion does not change appreciably with growth. However, enlargement of the frontal sinus continues in adulthood.

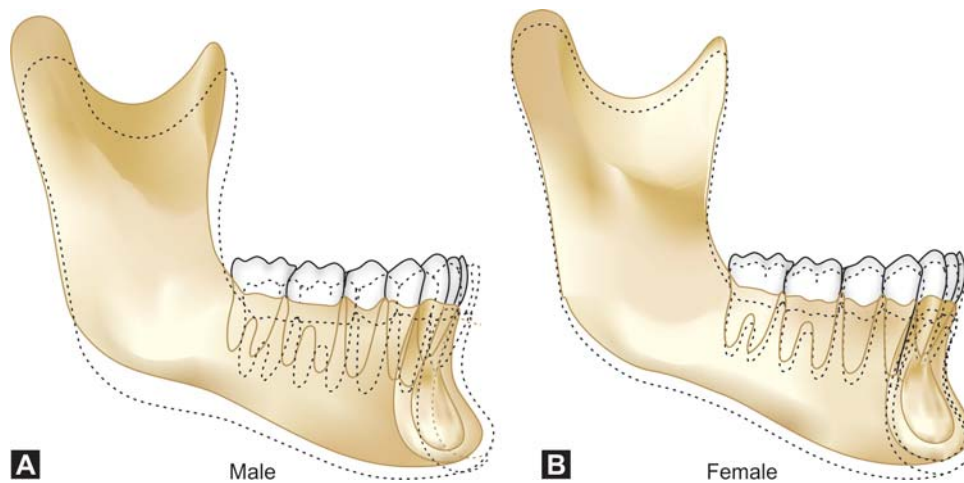
Midface: Downward movement of the posterior palate occurs leading to lowering of the palatal plane in the posterior area. Tip of the nasal bone elevates in adult growth, more in males than females. The key ridge and A point continue to move forward and downward with aging (Fig. 11.14).

Mandible: Mandible undergoes considerable change during aging. Gnathion, menton and pogonion move downwards and forwards with age more so in males than females. The changes in the B point suggest that the mandible is moving downward and forward with growth in males and females but with backward rotation

in females. The gonion and the anterior and posterior borders of ramus move downwards (Figs 11.15A and B).



Fig. 11.14: Maxillary dimensional and positional changes due to late growth



Figs 11.15A and B: Mandibular dimensional changes in male and female due to late growth

To Assess the Impact of Growth on the Orthodontic Treatment

Serial radiographs are evaluated to estimate increments in growth and subsequent effects of treatment. The four point superimposition of ricketts is used.

- *Cranial base superimposition:* The first superimposition point is along the basion-nasion plane at the superior aspect of pterygo-maxillary fissure.
- *Mandibular superimposition:* Internal mandibular structures are used such as outline of inferior alveolar canal and lingual surface of symphysis for superimposition.
- *Maxillary superimposition:* Internal maxillary structures are used for superimposition.
- *Maxillary displacement:* Superimposition is done along the basion-nasion line at nasion.

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Growth Prediction

CHAPTER OUTLINE

- Need for Growth Prediction
- Methods of Prediction
 - Hunterian concept
 - Gnomonic growth
 - Arcial growth by Ricketts
 - Moorrees mesh
 - Johnson's template
 - Todd's equation
 - Visualized treatment objective (Holdaway)
 - Visualized treatment objective (Ricketts)
 - Morphometrics methods

Variety is the spice of life. Every child extensively differs in the rate, amount and direction of growth and in the expression of the extent of his malocclusion. Dentofacial orthopedics is concerned with attempts to alter the size or direction or location of growth of some components of the craniofacial complex. Patients with class II malocclusion, for instance, may have only their class II molar relation in common but differ in their parameters of growth, timing of their pubertal growth spurt, the severity of class II relation of the jaws and their response to treatment. It is very difficult to predict the response to treatment especially when the patient is growing. Growth prediction helps in predicting to a certain extent the response to treatment and the growth changes. Growth assessment procedures indicate the growth status of the patient at a particular time and provide an assessment of the percentage of growth left. As Proffit says, growth prediction can never be accurate especially when the child is growing. *Growth prediction can be defined as the forecast of growth related changes with the objective of predicting the direction and amount of the growth of the maxilla and particularly the mandible*

as well as the timing of the adolescent growth period. Being able to predict growth will help craniofacial biologists in two principal ways: (i) The science of prediction demands that the knowledge of the craniofacial biologist be valid and cohesive and that they understand a great deal about craniofacial growth and (ii) Predicting the adult features of a patient will help in the interception and correction of malocclusion. In predicting craniofacial growth, two fundamental schools of thought exist. One school of thought uses the basic approach, seeking universal laws of growth applicable to humans, while the second group uses techniques for prediction which are based on histories, case examination, and radiographs, etc.

NEED FOR GROWTH PREDICTION

- Growth prediction helps the clinician to intercept and correct the malocclusion.
- Growth prediction can be used as patient education aids.
- Growth prediction (VTO) is helpful in 'visualizing' the treatment objectives and prioritizes the objectives, keeping in mind the growth pattern of the patient.
- Growth prediction is a tool for orthodontic treatment planning but without forcing any particular treatment procedure.
- Response to a particular treatment can be predicted, provided the patient continues in the same growth direction.
- If the patient continues to be in the growth phase even after treatment, growth following the conclusion of treatment can be predicted to plan for retention period, thus ensuring stability of the results.

METHODS OF PREDICTION

Prediction methods generally followed in science are of four types [Hirschfield 1971]. They are:

- Theoretical
- Regression
- Experiential
- Time series.

Growth Prediction in Orthodontics

Growth prediction is usually not done for all the cases but only for those cases in which perverted, altered growth pattern is seen. Ironically, predicting the altered growth pattern is the most difficult task, because every child has a genetically determined pattern which is acted upon by environmental influences. Growth prediction often does not satisfy the purpose it was originally invented for. Development of a satisfactory treatment plan is often contingent upon predicting the outcome of growth in the absence of treatment and anticipating the product of the interaction between the desired treatment objective and craniofacial growth. The failure to anticipate the effects of growth in a long range treatment plan can result in a marked deterioration of the desired outcome overtime. On the other hand, postponing treatment until the head has stopped growing might have disastrous consequences on the patient's self-image and ability to engage in social interactions during the period of the life cycle when peer relationships have a profound impact on the ability to form other relationships in later years.

The advent of cephalometric radiography, among its other frequent utilities, was actually for the study of growth related changes in the craniofacial complex. Early workers in the field quickly recognized the potential of lateral cephalograms in being used as tools to predict facial growth especially mandibular growth. Thus, in most of growth prediction methods be it the grid or the mesh diagram, radiographic cephalometry forms the basis around which the plot is constructed. Its easier now to classify growth prediction methods into (Box 12.1):

- Cephalometric methods.
- Non-cephalometric methods.

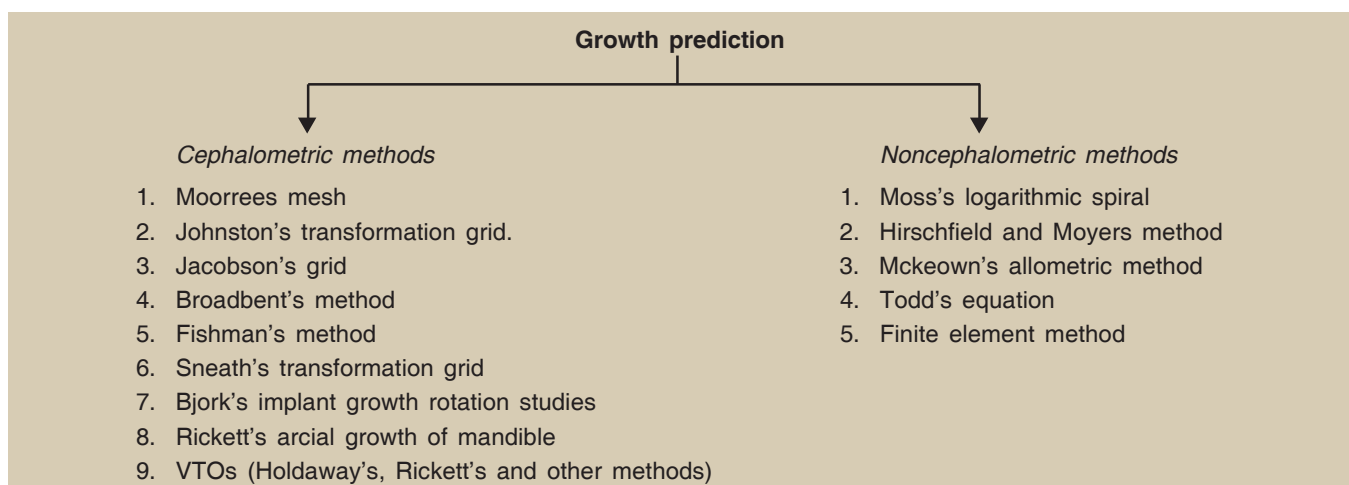
In Cephalometric methods, cephalograms form the basis for the prediction that is done in one or the other form of tracing using mesh or grids as reference. This is not to understand that noncephalometric methods do not use lateral cephalograms, they actually do, but mathematic formulas and allometric equations are used in the forefront to predict the growth.

Cephalometric growth prediction methods can further be classified as:

- *Template method*: The commonly used templates are: (i) Unisex Bolton template from ages 1 to 18 years; (ii) Burlington template, three configurations; (iii) Original Burlington or its Michigan modification; (iv) Johnston's template analysis.
- *Other cephalometric methods like VTOs*.

Growth prediction methods can be used as a short term forecast to plan strategy and anchorage and long-term forecast to give evaluation of final results and to

Box 12.1: Methods of growth prediction



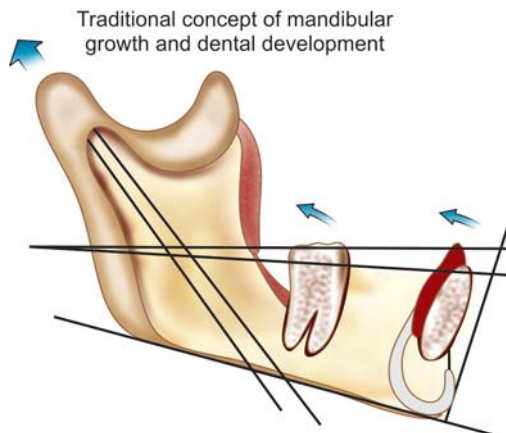


Fig. 12.1: Hunterian concept of mandibular growth

analyze esthetics and facial balance. It would be surprising to find out that most of the prediction methods, except a few, concentrate on the prediction of the growth of mandible more than on any other part of the face. Growth prediction, as applied to orthodontic practice is mainly the prediction of mandibular growth rather than the growth of the whole face, for example, in skeletal class II malocclusion, mandibular growth prediction forms the basis of treatment planning.

Hunterian Concept (Fig. 12.1)

John Hunter hypothesized a linear model for mandibular growth where there is resorption in the anterior border of ramus and deposition in the posterior border, thus lengthening the mandible. The Hunterian concept of mandibular growth was held as a dictum till Bjork proved that mandible and maxilla underwent rotational growth in his implant studies.

Gnomonic Growth and the Logarithmic Spiral

Following Bjork's growth rotation concept, Moss and Salentejin predicted that mandible's rotational growth was along a spiral path. Moss' method of predicting the mandibular growth along a logarithmic spiral derives its inspiration from the concept of "gnomonic growth" by D'Arcy Thompson. D'Arcy Thompson has explained the two fundamental features of chambered nautilus which is shown in Figure 12.2. (i) The shell grows in size and there is no change in shape. The original shape is maintained despite the asymmetric growth. This portion of increment, which even when added, does not alter

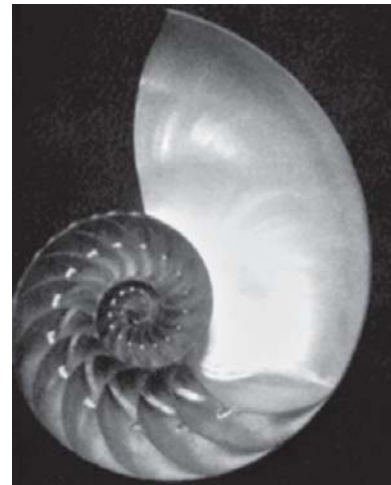


Fig. 12.2: A chambered nautilus

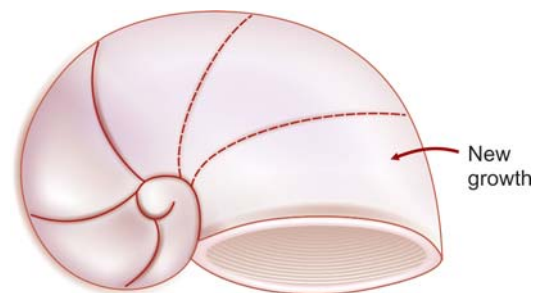


Fig. 12.3: Gnomonic growth of nautilus. There is increase in size but the shape is maintained

the shape but only produces an increase in size is called as *gnomon* in Greek by Aristotle (Fig. 12.3). The growth of the shell of a sea mollusk is along a logarithmic spiral, which is in perfect geometrical proportion. Thompson called the growth along the spiral as "gnomonic growth". As each chamber is added to the mollusk's body, the growth keeps in line not only with the spiral but also a perfectly increasing proportion. (ii) The next feature of the chambered nautilus is that the gnomonic growth can be described by a curve. This curve is called as *logarithmic or equiangular spiral* (Fig. 12.4). The important feature of this spiral is the movement of a point away from the pole along the radius vector with the velocity increasing with the distance from the pole. The angles formed with the pole will be equal. Using the algebraic equation $r = a^\theta$ can be written as $\log r = \theta a$ or since a is constant, $\theta = k \log r$. The formula implies that the vector angles about the pole are proportional to the logarithms of the

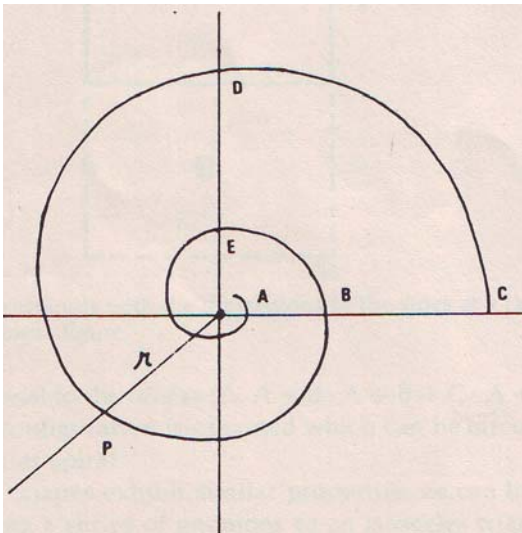


Fig. 12.4: Logarithmic spiral: r = radius,
 $\angle A = \angle B = \angle C = \angle D = \angle E$

successive radii. Hence they are called the logarithmic spiral.

Gnomonic Growth of Rectilinear Figures

A logarithmic spiral can be drawn from a rectangle whose sides conform to the golden ratio of 1:1.618. A square is drawn, inside this rectangle along the shorter of the lengths, i.e. parallel to the breadth of the rectangle and a quarter circle is drawn between the diagonally opposite corners of the square. A square and a quarter circle are drawn on the remaining side and the same is continued for all the remaining smaller rectangles inside the first rectangle. The joining of all the quarter circles will result in a spiral with logarithmic proportions.

The growth of the shell of a sea mollusk is along a logarithmic spiral, which is in perfect geometrical proportion. Thompson called the growth along the spiral as "gnomonic growth". As each chamber is added to the mollusk's body, the growth keeps in line not only with the spiral but also a perfectly increasing proportion. In the same manner logarithmic spiral can also be constructed from golden triangles. Thompson defines logarithmic spiral as follows: *Any plane curve proceeding from a fixed point or pole and such that the vectorial area of any sector is always a gnomon to the whole preceding figure, is called an equiangular, or logarithmic spiral.*

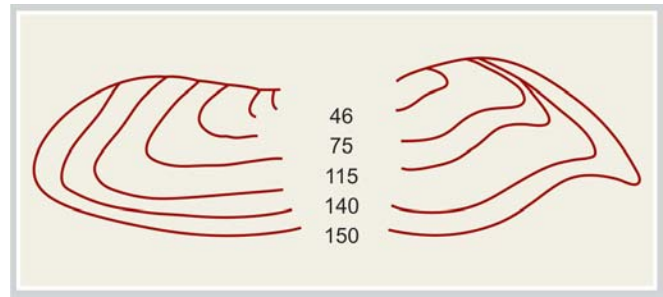


Fig. 12.5: Nasal (left) and Oral (right) functioning spaces of human fetuses demonstrating gnomonic growth (Adapted from: Moss and Salentijn. Morphological attributes of logarithmic growth of human face: Acta Anat 1971;78:185)

Gnomonic Growth of the Human Face

Moss after extensive study indicated that orofacial matrices manifest gnomonic growth. He sectioned the heads of human fetuses with crown-rump length ranging from 26 to 163 mm in the midsagittal section. Direct measurement of the oral, pharyngeal and nasal cavities demonstrated gnomonic growth of the nasal, oral and pharyngeal spaces (Fig. 12.5).

Moss and Salentijn further conducted a study on mandibles acquired at various ages from the skull of dead American Indians. Though being a cephalometric study, the annual growth of mandible was described using a logarithmic formula. Moss postulated that growth of mandible could be influenced by the inferior alveolar nerve and its direction depends on the course of the nerve. He called this 'the unloaded nerve' concept. Lead shots were implanted at three foramina along the path of the nerve, namely the foramen ovale, mandibular foramen, mental foramen. Lateral cephalograms of all the bones were taken. Mandibles at four different stages of development were included in the study: fetal, deciduous, mixed dentition and adult mandible. The X-rays were superimposed and it was found that the path of inferior alveolar nerve was along a logarithmic curve and that the four curves of all the mandibles were superimposed (Figs 12.6A and B).

The equation of the spiral is:

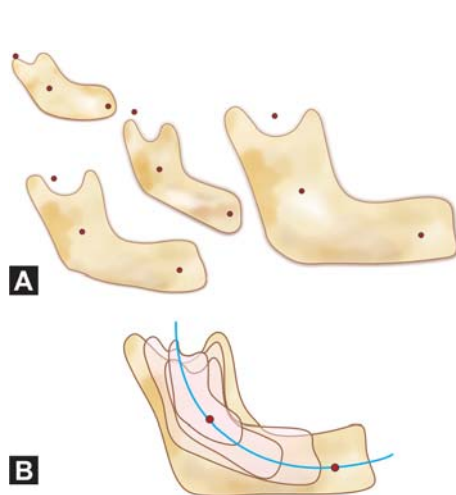
$$\log r = \log a + ko$$

where r = radius of the curve of the spiral

o = angulation of the radial line [in radians]

a = value of r when $o = 0$.

As the bone increases in size, the spiral itself does not change, instead, the mandible changes in position and



Figs 12.6A and B: (A) Location of foramen ovale, mandibular foramen, and mental foramen in the fetal, deciduous, mixed and adult skulls; (B) The foramina are aligned perfectly on a logarithmic spiral—a single curve

the base appears to rotate along the spiral, moving to a position where there is less curvature of a spiral because as the bone lengthens with growth, the distance between the foramina increases. During growth, the gonial angle that was obtuse becomes acute; due to clockwise rotation, the corpus remains horizontal and the position of ramus keeps changing and the mandible undergoes a clockwise rotation along the spiral path.

Arcial Growth of Mandible

The middle of 20th century saw some dramatic changes in the concept of mandibular growth. After Moss' postulate of logarithmic spiral, Ricketts described a pattern of arcial growth of mandible. The advantage of Ricketts arcial growth pattern was that an arc of growth can be constructed for every individual depending on the length of the core of the mandible. Improved visualization of the condyle and coronoid by laminography enabled Ricketts to better observe the bending of the mandible from infancy to maturity.

Principle of Arcial Growth

The normal human mandible grows by supero-anterior (vertical) apposition at the ramus on a curve or arch which is a segment formed from a circle. The radius of this circle is determined by using the distance from mental

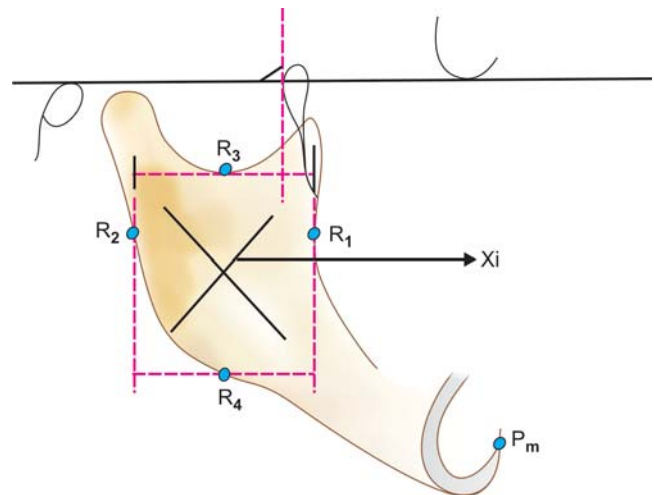


Fig. 12.7: Xi point is the geometric center of ramus. It is determined by the intersection of diagonals of a rectangle which has height and width of ramus as its sides

protuberance (Pm) to a point at the forking of the stress lines at the terminus of the oblique ridge on the medial side of the ramus (point Eva).

Landmarks (Figs 12.7 to 12.9)

Ricketts, in order to locate a central core which is immune to surface deposition and resorption, introduced the two important landmarks, his greatest achievement on the mandibular ramus, the Xi point and Eva.

- **Xi point:** It represents the geometric center of ramus (Fig. 12.7).
R₁: Most concave point on the anterior border of ramus.
R₃: Deepest point on the sigmoid notch.
R₂: Point opposite R₁ on the posterior border of ramus.
R₄: Point opposite to R₃ inferior border.

Xi point is a constructed point and is the intersection of diagonals of a rectangle with sides' tangent to deepest point on the sigmoid notch R₃ and most concave anterior border of ramus R₁. The other two sides are constructed as perpendiculars to the previous sides through the posterior border of ramus R₂ and the inferior border of ramus R₄. Xi point coincides with the opening of mandibular foramen/lingula. Surprisingly, in most of the cases, it is at the level of the occlusal plane of the patient. This gives

us an alternative method to construct Xi point, i.e. by locating the midpoint between R_1 and R_4 .

- *Eva*: Ricketts noticed that in the dried mandible, the stress lines run on the medial aspect parallel to the external oblique ridge and ascend up the ramus. The stress lines fork into two at a particular point. Ricketts named this point Eva to honor his mother. So Eva is the confluence or the point of the forking of the stress lines at the terminus of the oblique ridge on the medial surface of the ramus (Fig. 12.8).
- *DC point*: It is the midpoint or the bisecting point of the condylar neck.
- *PM point or Suprapogonion*: It is the point where the anterior border of symphysis of mandible turns from convex to concave. It was found to be the most stable point in mandible and is hence, used as reference.
- *MU point (Murray)*: This point was named after Ricketts' father. It is a point on the sigmoid notch where the curve from Eva touches the notch.
- *Tr/True radius*: It is the center of the arc. It is the intersection of arcs from Eva and PM drawn with Eva-PM as the radius.
- DC-Xi line constitutes the Condylar axis.
- Xi-PM line constitutes the Corpus axis.

The condylar and corpus axis together forms the core of mandible, the path of mandibular nerve.

Ricketts arcial growth of mandible was constructed using superimposition on serial lateral cephalograms and they were interpreted using computer analysis. Superimposing on corpus axis, registering at PM of serial cephalograms revealed a mandibular growth pattern in which the ramus was found to curve superiorly and anteriorly (Fig. 12.9). Using this premise, Ricketts constructed three arcs:

Curve A: Curve A was through DC-Xi and PM (Fig. 12.10). If mandibular growth had been along this curve, then it would open the gonial angle too wide, which normally does not happen. The resulting mandible will be too obtuse this way.

Curve B: This curve was constructed passing from the tip of coronoid process through the anterior border of ramus, and through Pm (Fig. 12.11). The mandibular growth along this curve would be bent too much.

Curve C (Fig. 12.12) Arc of Mandible: The final curve was between coronoid and condylar processes through

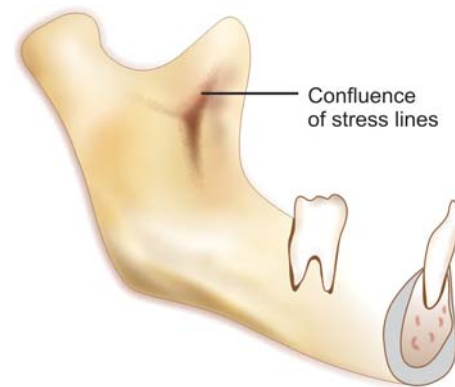


Fig. 12.8: Eva point represents the confluence of stress lines on the medial surface

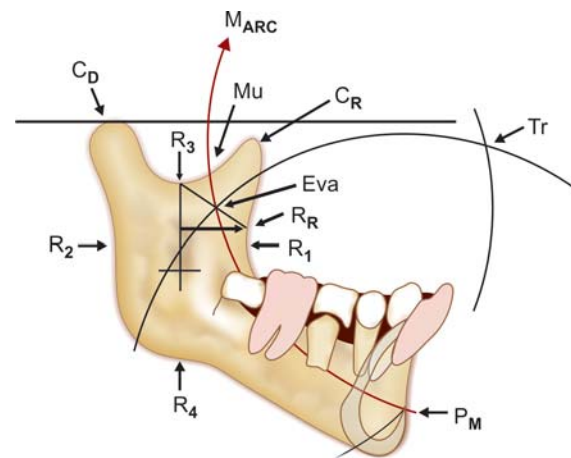


Fig. 12.9: Landmarks used by Ricketts

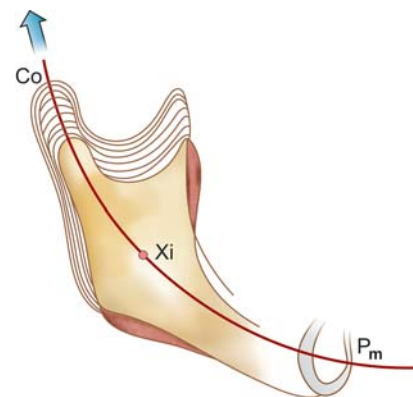


Fig. 12.10: Curve A

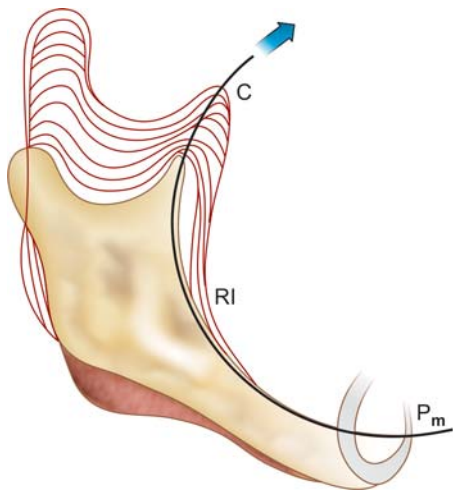


Fig. 12.11: Curve B

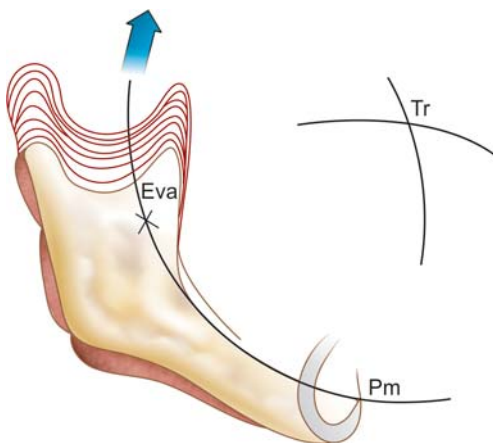


Fig. 12.12: Curve C. Tr represents the intersection of two arcs with their radii equal to Eva-Pm. Eva will form the center of one arc and Pm the other. The curve created through Eva and Pm with Tr as center represents the arc of mandible

Eva-PM. When annual increments of mandibular growth [about 2.5 mm] added and mandibular growth predicted along this curve, it was found to correlate very well with the final composite of the computer analysis. When a new point Tr is used as the center of a circle whose segment passes through Eva and Pm point and which has a radius equal to Eva-Pm, the true arc of the mandible was developed. Computer analysis revealed that the predicted mandible was absolutely correct in size and form when compared with the final composite.

Synthesis

With the arcial growth of the mandible, it is evident that mandibular growth is not linear but along an arc. Anterior resorption and posterior deposition pattern have been replaced by superior and anterior deposition of the ramus that takes place along a curve. The curve is a segment of a circle with the distance Eva-PM as the radius. Bending of the mandible was occurring in an orderly manner. greater the magnitude of growth, greater is the bending. Form and size of mandible was predicted after 5 years with first cephalogram as reference and compared with the X-ray taken after 5 years. The coronoid and condylar processes grow upward and outward in the direction, essentially as a function of the curve of original arc. sigmoid notch growing in arcs of small radius stayed small and arcs of wider radii had coronoid and condylar processes diverging apart. Gonial angle drifted posteriorly on the arc almost one half the total increases in mandibular growth on the arc. Annual increase of 2.5 mm was observed in mandible and growth was found to cease at 14.5 years for females and 19 years for males. Conceptual framework about the direction of mandibular growth has shifted from being horizontal to vertical with Ricketts' arcial growth.

Occlusal plane and mandibular teeth eruption: As mandible grows along the arc, space is created for the 2nd and 3rd molars. According to Ricketts, the anterior border does not have to undergo resorption to accommodate the molars because the molars erupt upward and forward. With arcial growth and upward and forward eruption of teeth, the chin is pushed beneath the lower arch (Fig. 12.13).

- The angle of the occlusal plane to corpus axis remains constant.
- The biologic relation to the development of the posterior end of occlusal plane to Xi point - Distal end of occlusal plane drops down during growth.
- Relation of the lower lip to the occlusal plane remains constant without treatment. As the growth of the arc continues, symphysis, or chin is pushed under the denture as the teeth erupted upward and forward. This will explain the chin button development.
- The anteroposterior movement of lower incisor is biologically related to A-Pg plane. Prediction of the anterior position of the lower incisor related to the prediction of change in convexity.

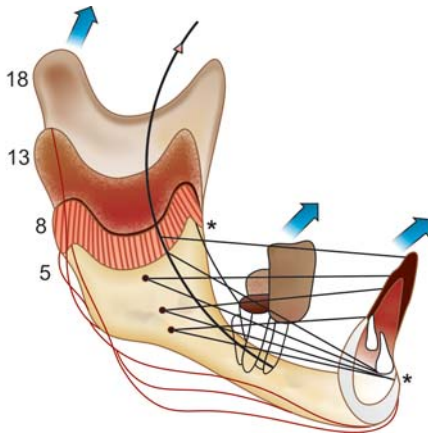


Fig. 12.13: Serial tracings showing relation of teeth eruption and arc of mandible

- The lower molar erupts upward and forward with the occlusal plane. This creates space for the third molar. If the molars are restrained from moving forward, there may be space loss for the 3rd molar.

Moorrees Mesh

Moorrees constructed a template in the form of a mesh which was used to superimpose growth changes in serial lateral cephalometric radiograph. Before Moorrees, the study of human face using grid system was performed by Leonardo Da Vinci as early as 1490. More contemporary workers like De Coster also advocated the use of transformation of a mesh co-ordinate system in 1939. The concept was taken up and developed by Moorrees.

Construction (Fig. 12.14)

Moorrees mesh consists of equal sized rectangles about 24 in number, the landmarks are correlated to the specific position of landmarks in the mesh. The grid consists of a core rectangle, the size of which varies for every patient and this determines the size of all the other rectangles. The reference lines are extracranial vertical lines, to start with, a vertical parallel to extracranial vertical is drawn through nasion. Two horizontal lines are drawn perpendicular to the extracranial vertical, one through nasion and the other through ANS. The last vertical is drawn parallel to vertical through nasion at a distance of NS from nasion. This forms the core rectangle which is divided equally into four by two lines. The length and

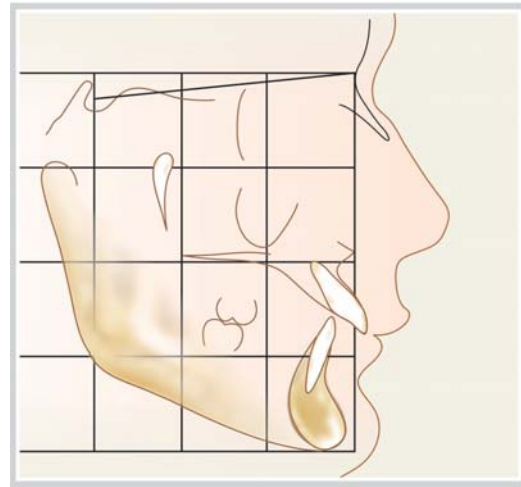


Fig. 12.14: Analysis of facial forms using mesh

breadth of the smaller rectangles are taken as reference to draw the remaining 20 rectangles. This is achieved by drawing verticals, one in front and one behind the core rectangle at a distance equal to the length of the smaller rectangle. Horizontal lines, one above the core rectangle and three below it are drawn at a distance equal to the breadth of the smaller rectangle. Now the face is inside a grid system formed of series of rectangles.

Vertical lines are numbered from 1 to 5 and horizontal lines from A to G. Every line in the grid corresponds to position of specific landmark and when that landmark is changed in position, the lines are distorted. The list of landmarks are given in Tables 12.1 and 12.2, the change in position of them distorts the respective lines.

Relating a patient's face sagittally and vertically to the mesh diagram depends on two dimensions, namely (i) length of the anterior cranial base and (ii) upper facial height [nasion to ANS along the vertical].

The proportional horizontal and vertical location of each landmark with respect to that in the norm is computed. The first step is the assessment of an individual in comparison with the size of rectangles. Smaller faces have smaller core rectangles and vice versa. There is enormous inter-individual variation. The size increases from 8 to 16 years of age, height increments are 4.5 mm for boys and 3.5 mm for girls and length increments are 3.2 mm for boys and 2.4 mm for girls.

The change in position of each landmark at a particular age period [8-16 years] is calculated as a percentage shift. There is usually an increase in overall size of the patient's face; every landmark undergoes

Table 12.1: Vertical lines

Vertical lines	Landmarks
1.	Glabella, soft tissue nasion, tip of nose, subnasale, labrale superius, stomion, labrale inferius, supramentale, soft tissue pogonion.
2.	Bony landmark: Nasion, ANS, point A, point B, incisal edges of maxillary and mandibular incisors, pogonion, gnathion.
3.	Distorted last because it is influenced by lines 2 and 4. PTM, PNS, posterior extent of occlusal plane.
4.	Articulare, Basion, Gonion.
5.	Follows vertical line 4

Table 12.2: Horizontal lines

Horizontal lines	Landmark
A	Distorted for the vertical location of sella turcica.
B	Distorted for the vertical location of sella turcica.
C	Tip of nose, articulare, Basion.
D	Tip of nose, PNS, Articulare, Basion, passes through ANS.
E	Stomion, incisal edges of upper and lower incisor, gonion, occlusal plane.
F	Gonion, gnathion.
G	Follows line F

growth/change by a certain extent with large inter-individual difference but the facial type remains constant through 8 to 16 years. Retrognathic mandibles have posterior distortion of the rectangles; while high mandibular angles will have downward displacement of the mesh.

Johnston's Grid

Johnston designed sex specific templates by utilizing the numerical standards obtained from the publications of Riolo et al who used the cephalograms from University of Michigan. The subjects were untreated girls and boys with normal occlusion and class I and II malocclusion. Johnston's grid though appearing very complex, is actually extremely simple when used. Johnston's template uses normative standard [by including children with malocclusion] rather than ideal standards which can be difficult for comparison while using for children with altered growth.

To describe the template in a very simple way, they are age specific. The use of values like angles in degrees and millimeter linear measurement is unwarranted. Each sex specific template [male and female] has an oriented set of rulers which are graded in years, from 6-16 years [in even numbers], thus they are age-specific. Growth can be analyzed for children in this age group directly from the template.

In the first step, the overall growth of the facial skeleton is assessed which is followed by the second step where isolated areas of growth discrepancy are assessed.

Method: The Johnston's forecast grid shows the average increments of growth per year for the points nasion, A, B, nose and posterior nasal spine. It also gives a method of constructing pogonion, given a B point. There is no individualization in this method, in that, all the patients will grow the same amount horizontally and vertically irrespective of their facial patterns. This method is accurate to about 70 percent (Fig. 12.15).

Cranial Base Superimposition: Maxillary and mandibular growth are always related by holding a third plane of reference, either cranial base or FH plane. Cranial base generally used is SN plane; other planes Ba-N, PMV. SN plane, registered at S is the easiest plane to use, because the landmarks are easily located by a common practitioner.

FH plane registered at PTV has been advocated by Johnston because he believes it is closer to the jaws but, practically speaking, the porion and orbitale points are difficult to locate. The cephalogram of the child under study is superimposed on the template over SN registered at S. The superimposition will show us the position of all the other structures like the maxilla, mandible, position of upper and lower incisors, points A and B, pogonion, etc. (Fig. 12.16). The skeletal age in years of the child

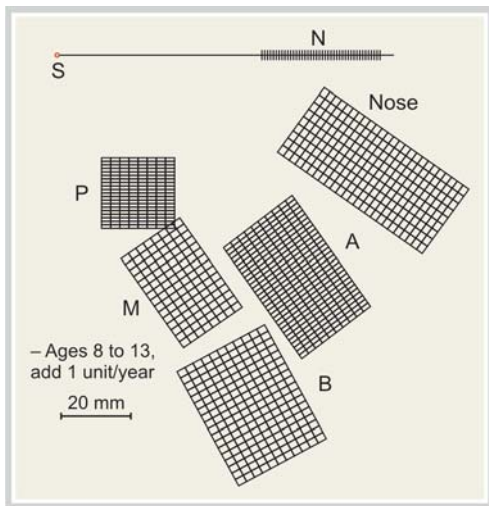


Fig. 12.15: Forecast grid developed by Johnston. S, sella; N, nasion; P, posterior nasal spine; Nose, tip of nose; M, any point on the crown of maxillary first molars; A and B, subspinale and supramentale. The tracing of the landmarks is superimposed along S-N and registered at S. The points are then advanced downwards one unit per year

is directly studied from the template by noting the correlation of every structure (maxilla, mandible, etc.) with the rulers on the grid. Individual variations in skeletal structures are detected at this point, namely, maxilla lagging behind mandibular growth.

Regional Superimposition: Regional template is placed over the cephalogram and measurement is made between 2 points [e.g. PNS-ANS for maxilla, GO-Pg for mandible] and age noted in years (Fig. 12.17).

Prediction: Every part of the skeletal framework is in the form of a grid, e.g. maxilla, mandible. The grid system (forecast grid) was constructed on the assumption that the growth changes are regular annual changes and direction of growth is average. To predict facial growth, every point is advanced one grid unit per year superimposed on SN registered at S.

Uses of Template:

- The relative age of the skeleton is measured, for example, a patient who is 11 years old can have an overall skeletal age of 9 years [cranial base, maxilla and mandible] which is normal but if the cranial base and maxilla are uniformly 11 years and mandible alone is 8 years, this condition is abnormal. Thus the mandible for that age is small.

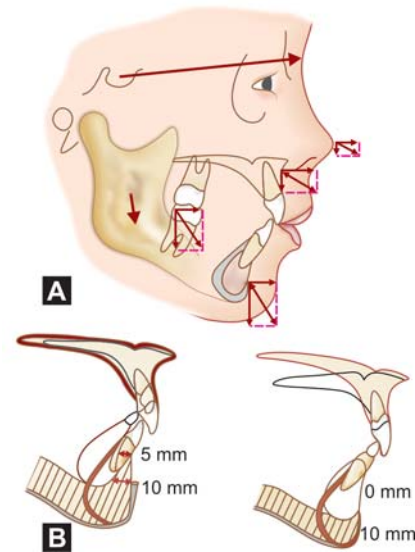


Fig. 12.16: (A) Johnston's method of prediction. Each point is extended to predict the average amount and direction. The basic reference line is sella-nasion. (B) Comparison of point B growth with pogonion in both horizontal and vertical directions

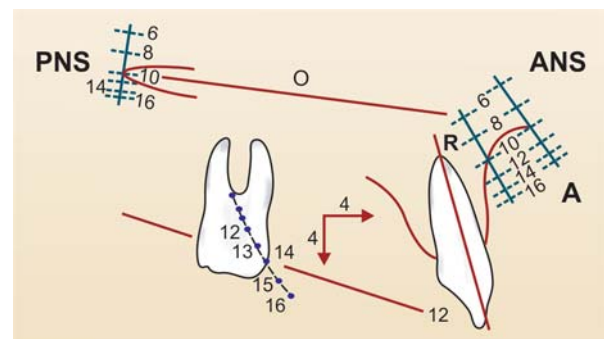


Fig. 12.17: Regional superimposition

- Angles of cranial base, gonial angle, mandibular, occlusal plane angles, and palatal plane angle can be assessed and vertical growth of the face is assessed.
- Position of upper and lower incisors can be directly seen, the differentiation between proclination and forwardly positioned incisors is easily made out.
- Vertical facial proportions are judged in years; AFH 10 years but PFH only 8 years.
- Malocclusion is the integration of small deviations of all parts of the face. Deviation from normal of every part can be individually assessed, i.e. to say that dental

or skeletal cause of malocclusion is differentiated, for example, skeleton is normal but maxillary dentition has moved forward.

Todd's Equation

The disadvantages of ordinary prediction methods are:

- They are all in the form one of linear coordinate system, either rectangle or grid system, it cannot predict radial growth, the angular coordinate of each landmark remains a constant.
- There are many frames of reference used to describe growth. Every frame of reference can give a description of growth changes, for example, SN superimposition is markedly different from FH plane superimposition.

All the growth prediction methods described changes in the craniofacial morphology as an ordered phenomenon. Though growth follows an inherent trait and has a genetic influence, the external environmental forces on growing objects cannot be ignored. Todd considered that biomechanical influences on growth and the foremost influence is that of the force of gravity.

One of the most obvious global influences on the biomechanics of growth is the force of gravity. This force is exerted on every point within the craniofacial complex, and it also provides a counter force for the action of muscles. In order to appreciate the overall regularity of gravitational pressure, it is useful to consider the human skull as a spherical tank filled with fluid. From elementary hydrostatics, we know that the amount of pressure (P) at any point (R, θ) on the surface of the tank is uniquely determined by its vertical distance from the top of the sphere (Fig. 12.18). The direction of pressure is normal (perpendicular) to the surface at every point, and the amount of pressure can be expressed as a function of position by the following equation:

$$P = a R (1 - \cos \theta)$$

where 'a' is a constant representing the product of the force of gravity and the density of the fluid. Thus, if all bone elements were placed or displaced in the direction of gravitational pressure, as suggested by Wolff's law, then they would all move outward along radial lines emanating from the center of the sphere. Moreover, if changes in mass reflect the amount of gravitational pressure, then the displacement along these radial lines of growth would increase monotonically over time as a function of pressure. If both of these assumptions are viable, then the overall

pattern of change can easily be expressed in polar coordinates with a single pair of equations:

$$\theta' = \theta$$

$$R' = R + b P$$

where b is an increasing function of time. The overall effect of this transformation on a human facial profile is depicted in Figure 12.19. Not only does it create a perceptual impression of human growth, but it is also consistent with the data obtained through radiographic cephalometry.

This analysis, of course, is oversimplified. Heads are not perfectly spherical, there are other sources of stress operating on the craniofacial complex besides the force of gravity, and the relative orientation of the head with

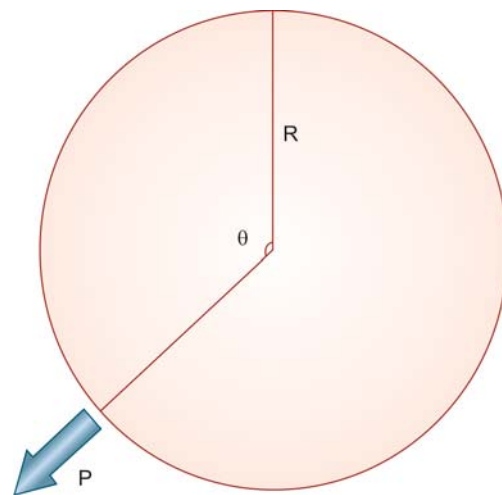


Fig. 12.18: Pressure distribution inside a fluid filled tank. The amount of pressure (P) at any points (R, θ) on the surface is determined by its vertical distance from the top of the sphere

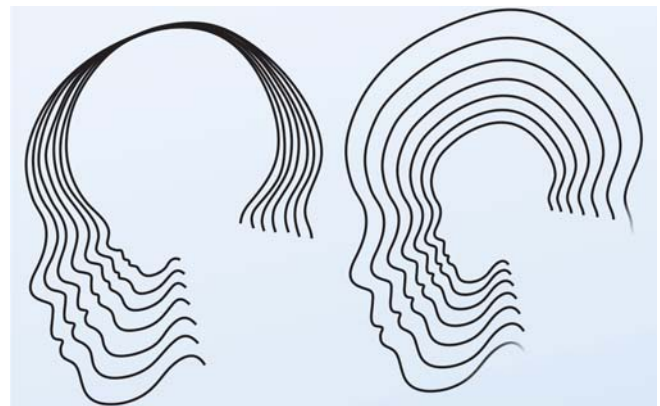


Fig. 12.19: Growth of human head in sequence

respect to gravity does not remain absolutely fixed. The resulting model should be thought of as a kind of ideal case, similar to analyzing the motion of a falling body without considering the air resistance. Such a model can be quite useful if it helps us to appreciate the global influences on craniofacial growth or provides a means of approximating the course of growth in any given individual.

Predicting craniofacial growth: To test the equation, lateral cephalogram of subjects taken at younger (T1) and older (T2) groups were taken. The initial T1 cephalogram was traced and ANS and Gn were located and traced. Superimposition was done on polar graph paper so that ANS and Gn were at 125° and 160° radial coordinate. The second cephalogram T2 was placed over young cephalogram and centered so that:

- Tips of the heads [vertex] were superimposed.
- ANS and Gn in older profile were as near to polar co-ordinates as in the younger cephalograms.

The profile of the patient is marked as series of points 3 to 5 mm apart (arbitrary point) by using the equations, a continuum of skull outlines were drawn. The predicted skull outline is compared with profile at older age. The mathematical transformation, which was derived from consideration of the global stresses on an idealized human head, is shown to make reasonably accurate growth predictions over a span of about 10 to 15 years. This finding is very important because the transformation changes both shape and size of the profile and the transformed profile does not have to be normalized for size with respect to the actual profile resulting from growth. The predictions that were made with this growth model were not totally accurate because of the mechanical sources of error and, perhaps, oral habits, such as thumbsucking, nailbiting, mouthbreathing, teeth-clenching, or unusual facial expressions or facial postures. Nevertheless, they very closely predict the actual outcome of growth. The growth rate in different subjects and within the same subject, and the direction of growth usually follow a predicted path if not hampered by other factors like trauma, illness, etc. The use of such a global transformation for this purpose may add an important dimension to treatment planning because facial profiles are represented as a continuous contour rather than as a collection of independent points.

Visualized Treatment Objective (VTO)

Visualized Treatment Objective was coined by Holdaway. A VTO is a *cephalometric tracing representing the changes that are expected during treatment* (Proffit). Ricketts defines VTO as a visual plan to forecast the normal growth of the patient and anticipated influences of treatment, to establish individual objectives that are to be achieved for that patient. VTO is a relatively simple and accurate method of predicting the molars and incisor relations on the basis of growth and treatment alterations in the dentoskeletal framework.

Jacobson and Sadowsky (1980), have outlined the accomplishments of VTO as follows:

- It predicts growth over an estimated treatment time, based on individual morphogenetic pattern.
- Analyses the soft tissue profile.
- Graphically plans the best soft tissue profile for every patient.
- Determines favorable incisor repositioning, based on an ideal projected soft tissue facial profile.
- Assists in determining the total arch length discrepancy when taking into account 'cephalometric correction'.
- Aids in determining whether the case is extraction or non-extraction.
- Aids in deciding which tooth to extract in an extraction case.
- Aids in planning treatment mechanics.
- Aids in deciding which cases are more suited to surgical and/or surgical orthodontic correction.
- It provides a visual goal or objective to be achieved at the end of treatment.

Advantages of VTO:

- Establishment of specific treatment goals.
- Formulation of specific treatment plan to attain the treatment goals.
- Allowing rapid comparison of different treatment options before arriving at a final treatment plan.
- Assists in measuring and monitoring treatment progress, for making mid-treatment correction.
- Enhancing communication between patients, parents and clinicians.
- Etiology for variation in treatment response can be recognized like lack of patient cooperation, variation in growth pattern, etc.

Limitations of VTO:

- Use of average growth increments in prediction.
- Use of existing morphological traits to predict future events.
- VTO is presented as an exact representation of treatment outcome which cannot be so in all the cases.

Holdaway's VTO

Holdaway's VTO has 12 sequential steps. The plane of reference is SN plane.

Step 1: Place a clean sheet of tracing material over the original tracing, copying (1) the frontonasal area, both hard- and soft-tissue, with the soft-tissue nose carried down to near the point where the outline of the nose starts to change directions; (2) the sella-nasion line; and (3) the nasion-point A line (Fig. 12.20).

Step 2: First, superimpose on the SN line and move the tracing to show expected growth (0.66 to 0.75 mm per year unless a pubertal growth spurt is expected from wrist plate studies). Second, copy the outline of sella. Third, either copy or change the facial axis (Ricketts' foramen rotundum to gnathion) as you expect it to behave according to the facial type of the patient and the treatment mechanics that you customarily use in such

cases (The facial axis line is usually opened about 1° , but it may even be closed if one is confident that mandibular growth of the forward rotational type will occur during treatment) (Fig. 12.21).

Step 3: *Vertical growth of mandible, determining anterior facial height:* Superimpose the VTO facial axis on the original and move the VTO up so that the VTO SN line is above the original SN. The amount of movement will usually be 3 mm per year of growth, except in accelerated growth-spurt periods (*Note:* Since the facial axis may be opened or closed as judged from the facial pattern, the SN lines will not be parallel if we have changed the facial axis). Second, copy the anterior portion of the mandible, including the symphysis and anterior half of the lower border. Also draw the soft-tissue chin, eliminating any hypertonicity evident in the mentalis area (slightly round out this area). Third, copy the Down's mandibular plane (Fig. 12.22).

Step 4: *Anteroposterior growth of mandible, determining the posterior border:* Superimpose on the mandibular plane and move the VTO forward until the original sella and the VTO sella are in a vertical relation. Next, with

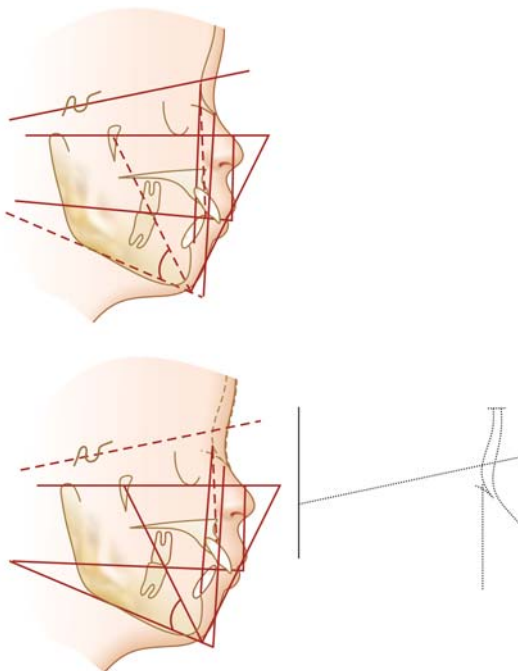


Fig. 12.20: Step 1 of Holdaway's VTO

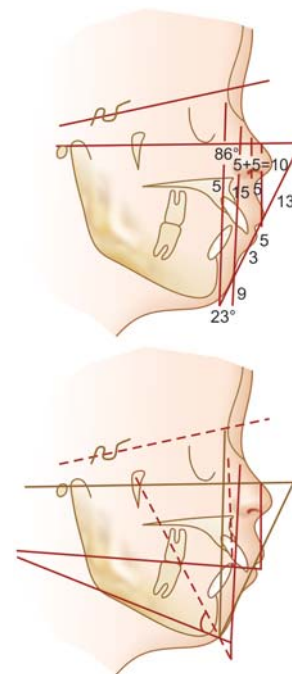


Fig. 12.21: Step 2 of Holdaway's VTO. Express growth in the frontonasal area for the estimated treatment time. Here horizontal growth is expressed in the frontonasal area for the estimated treatment time

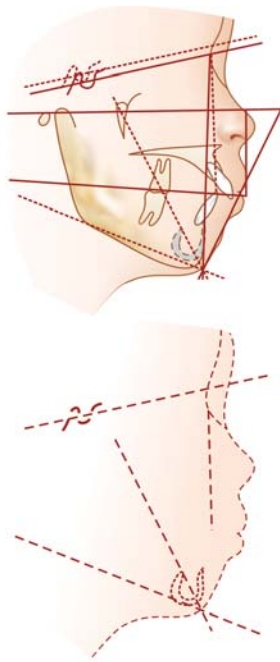


Fig. 12.22: Step 3. Express growth of the mandible in its vertical and anterior growth pattern and draw the anterior portion of the mandible, the soft-tissue chin, and the Downs lower border of the mandible line

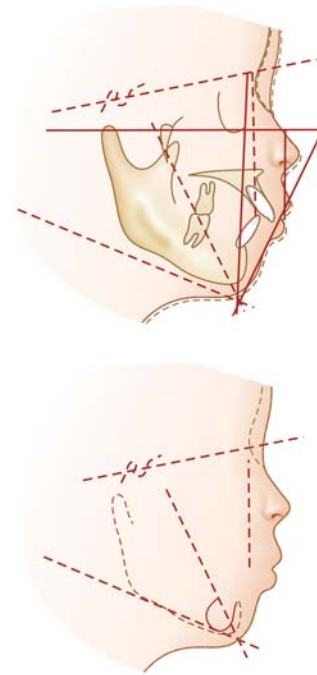


Fig. 12.23: Step 4. Express growth in a horizontal direction in the mandible (or lower face) and draw the posterior portion of the mandible

the tracing in this position, copy the gonial angle, the posterior border, and the ramus.

Finally, superimpose on sella to complete the condyle. Note: At this point, total vertical height has been forecast, as has the forward location of the chin structures, both hard and soft, and consideration will have been given to the effects of treatment mechanics on vertical dimension. One should not open the facial axis more than 1° to 2° because greater opening than this is usually inconsistent with good treatment mechanics (Fig. 12.23).

Step 5: Maxilla and lower nose: First, superimpose the VTO NA line on the original NA line and move the VTO up until 40 percent of the total growth is expressed above the SN line and 60 percent below the mandible (Note: This may be varied as you perceive the facial type to be short or long).

Second, with the tracing in this position, copy the maxilla to include the posterior two-thirds of the hard palate, PNS to ANS to 3 mm below ANS.

Third, also with the tracing in this same position, complete the nose outline around the tip to the middle of the inferior surface (Fig. 12.24).

The vertical growth of the nose over the usual 18 to 24 months of estimated treatment time keeps pace with the growth from the maxilla vertically to the anterior cranial base. Thus, its relationship to ANS is relatively constant. In some cases, there may be an elevation of the nasal bone and greater development of the nasal bulk, but this is difficult to predict and thus some noses will have changed form more than this VTO procedure suggests.

Step 6: Occlusal plane: With the VTO still superimposed on the line NA, move the VTO so that the vertical growth between the maxilla and the mandible is expressed 50 percent above the maxilla and 50 percent below the mandible. Second, with the tracing in this position, copy the occlusal plane. It is 3 mm above the lip embrasure (Fig. 12.25).

Step 7: Lip contour: The harmony line is drawn tangent to the lower chin. Upper sulcus depth on the average range 3–7 mm, with mean of 5 mm. The angle of harmony line is adjusted so that the upper sulcus depth is 3–3.5 mm. Superimposition is made on maxilla, NA line and occlusal plane, upper lip is drawn. To draw the

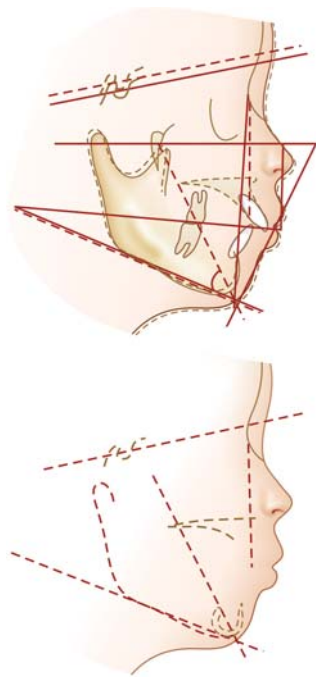


Fig. 12.24: Step 5. Locate and draw the maxilla, the new A point, and the lower part of the nose

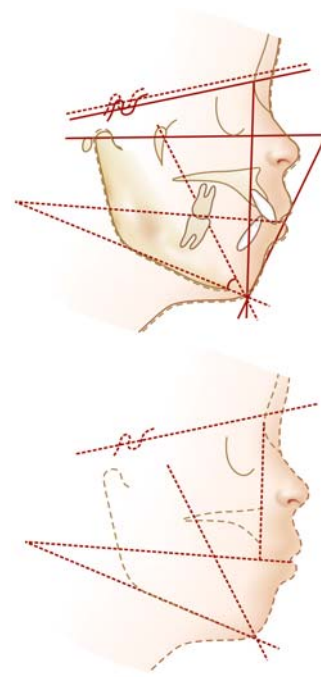


Fig. 12.25: Step 6. Occlusal plane location

upper lip, a template can be used. The lower lip is drawn touching the lower lip or 1 mm in front of it. Lip template by Jacobson and Sadowsky can be used to decide the lip contour (Fig. 12.26).

Step 8: Upper Incisor Position: The upper incisor position is determined by:

- Lip strain.
 - Upper lip change.
 - Maxillary incisor rebound.
- a. Lip strain is measured as the difference in thickness between basic lip thickness and thickness of the vermillion border. If the vermillion border is thinner than the lip thickness, it is then calculated as the lip strain. For example, if lip thickness is 16 mm and vermillion border is 10 mm, then lip strain is 5 mm; leaving a leeway of 1 mm.
 - b. The change in position of the upper lip from pretreatment to VTO is calculated.
 - c. There may be a rebound tendency for maxillary incisor by 1.5 mm; it is added to the previous measurement.

The superimposition is again carried out on the NA line and maxilla, and then the maxillary incisor is drawn taking into account the above mentioned criteria, viz.

change in axial inclination of upper incisor, relation of upper incisor with the occlusal plane (Fig. 12.27).

Step 9: Lower Incisor position: Lower incisor is drawn in harmonious relation to upper incisor with superimposition on the mandibular plane and symphysis with occlusal plane as the guide. The lower incisor is tipped back about the apex unless bodily movement is desired. The number of millimeters of lower incisor movement is noted and discrepancy is calculated. To find the arch length loss, the value is multiplied by two and the amount of crowding is added (Fig. 12.28).

Total discrepancy = $(2 \times \text{lower incisor movement}) + \text{crowding (calculated by model analysis)}$.

Step 10: Lower molar position: Position of the lower molar is determined after the position of lower incisor is drawn. In extraction cases, lower molar is moved forward for the remaining amount. In minimum discrepancy cases, lower molar is tipped back to find out whether non-extraction treatment can be done (Fig. 12.29).

Step 11: Upper molar position: With occlusal plane and lower molar as guide, upper molar is drawn in class I relation to lower molar. Amount of upper molar

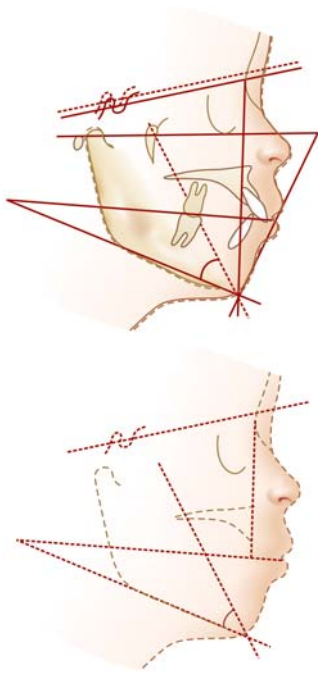


Fig. 12.26: Step 7. New H or harmony line is drawn and, using it as a guide, draw the most ideal lip position and form possible for that patient

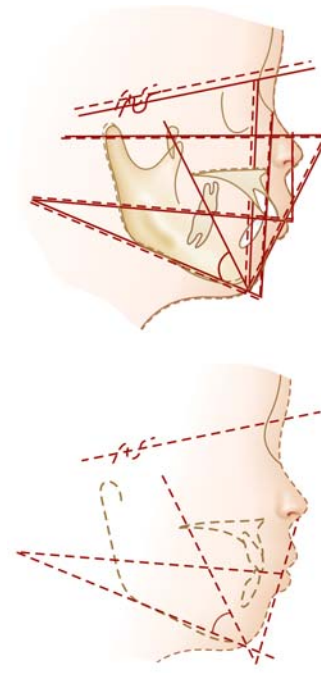


Fig. 12.28: Step 9. Reposition the lower incisor and calculate the effect of this on lower arch length

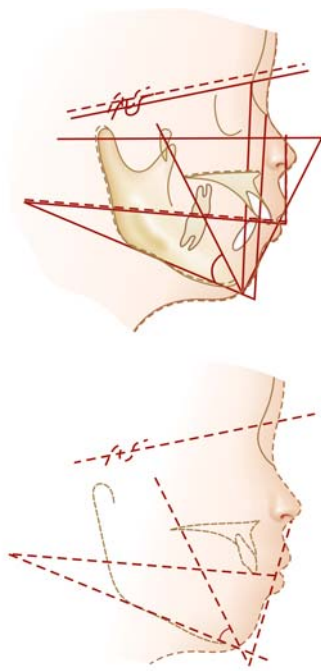


Fig. 12.27: Step 8. Maxillary central incisor relocation

movement is calculated by keeping the original NA line and maxilla as a guide (Fig. 12.30A).

Step 12: Point A: The position of point A is assessed by the best fit of maxilla. Change in position will be drastic for bodily movement of upper incisor and orthopedic appliances. The change in position of point A is analyzed and drawn (Fig. 12.30B).

Ricketts' VTO

Ricketts constructed VTO based on specific areas. According to Ricketts, growth changes of the craniofacial complex should be studied by keeping the center of least growth as the registration point. From various studies, Ricketts found that the center of least growth is near the pterygomaxillary fissure because growth changes occur in a radial manner from this area while the area itself remains constant (Fig. 12.31). He constructed a point at the intersection of basion-nasion line to the facial axis and named it the 'cc point' and then decided that his superimposition will be on Ba-N plane registered at cc point. He divides areas to be predicted into six:

1. Cranial base prediction.
2. Mandibular growth prediction.

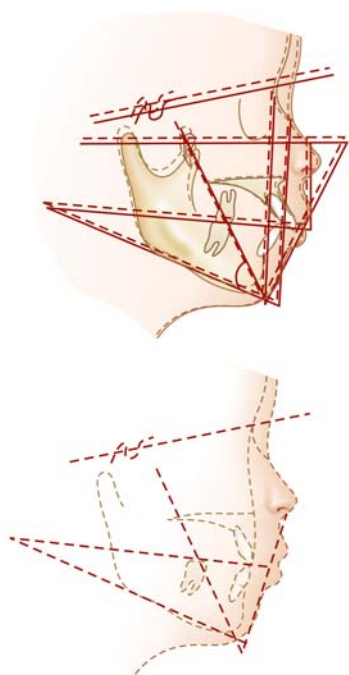
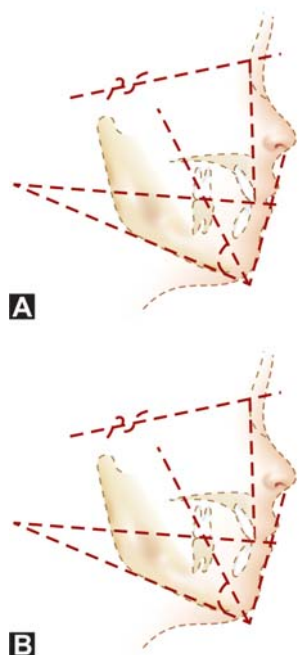


Fig. 12.29: Step 10. Determine the lower first molar position, considering total arch length discrepancy



Figs 12.30A and B: (A) Step 11. Reposition the maxillary first molar. (B) Step 12. Complete the artwork in the area involving point A, in the anterior portion of the hard palate, and in the lower alveolus lingually and labially

3. Maxillary growth prediction.
4. Occlusal plane position.
5. Location of dentition.
6. Soft tissue of the face.

Cranial Base

Place a tracing over the original tracing, superimposing on Ba-N and registering at cc, grow nasion 1 mm/year for two years (generally VTO are done for two years keeping in mind the orthodontic treatment period). Similarly, basion is also grown 1 mm/year. Slide to coincide old and new nasion and trace nasion area, and similarly trace the basion area.

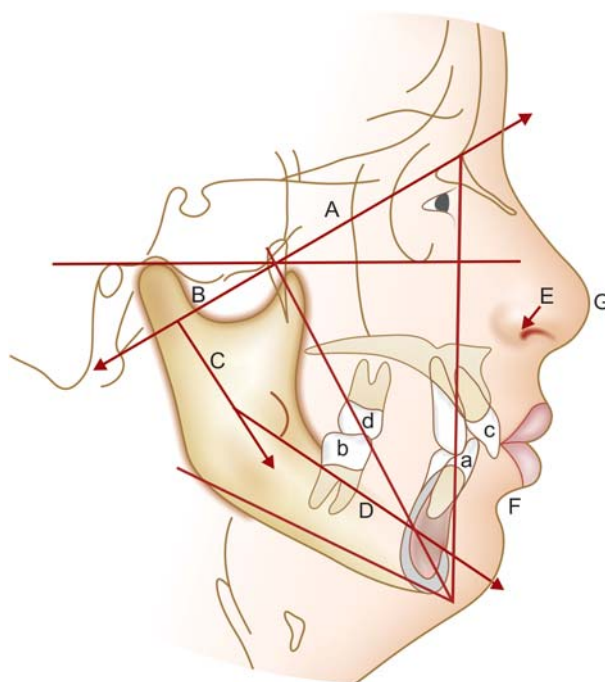


Fig. 12.31: Steps and sequences in short route procedure of forecasting. Anterior and posterior increases are estimated A = 0.8 mm; B = 0.8 mm; C = 0.8 mm; D = 1.6 mm (double the C)

E = Elective as decided by operator

F = Soft tissue is dependent on tooth movements

G = Dependent upon ANS and is 1.0 mm yearly.

Teeth Sequence:

a—lower incisor is + 1.0 mm to APo with CD = + 2.0 mm.

b—lower molar as determined by anchorage needs of d c.

c—upper incisor change as needed from lower incisor.

d—upper molar = 3.0 mm distal to lower in normal occlusion.

Mandibular Growth Prediction

The mandibular angle changes for every treatment procedure. It is calculated as degree of opening or closing of facial axis. The facial axis opens 1° with growth. The following are changes in the facial axis:

Convexity reduction—facial axis opens $1^\circ/5$ mm.

Molar correction—facial axis opens $1^\circ/3$ mm.

Overbite reduction—facial axis opens $1^\circ/4$ mm.

Cross bite correction—facial axis opens $1^\circ-1\frac{1}{2}^\circ$, recovers half the distance.

Facial pattern—with dolichofacial pattern the angle opens 1° and vice versa.

- Mark DC point, superimpose on Ba-N plane and register at basion. The line Ba-N is rotated up at the nasion to open the mandibular plane/bite and down at the nasion to close the bite using DC as fulcrum.
- Draw condylar axis, coronoid process and condyle.
- To predict the growth of condyle, mark points approximately 1 mm/year down the DC point on the condylar axis.
- Slide the tracing along the condylar axis so that the new mark is on Ba-N plane. Extend condylar axis to Xi point. Mark new Xi point.
- Now corpus axis is drawn by superimposing old and new Xi point, the increments per annum are 2 mm for PM.
- After new PM point is marked, trace the posterior border of ramus and lower border of mandible.
- Slide back along the corpus axis, superimposing old and new PM, trace symphysis and mandibular plane.
- Draw the facial plane with new nasion and pogonion.
- Facial axis is extended to the new Gn [where N-Pg and mandibular plane cross].

Maxillary Growth Prediction

Now the height of the anterior and posterior face has been determined. Maxilla's new position has to be predicted within this vertical dimension. The distance between old and new menton is divided into three portions (thirds) by distinct marks and named 1, 2 and 3. Superimposing mark 1 on the original menton along the facial plane, trace the palate. Point A is not traced at this juncture because it undergoes extensive changes with growth and treatment. With normal growth, point A should move forward but it undergoes the changes with treatment as mentioned in Table 12.3.

Table 12.3: Changes observed at point A following various treatment procedures

• Headgear	–8 mm
• Class II elastics	–3 mm
• Activator	–2 mm
• Torque	–1 to –2 mm
• Class III elastics	+2 to +3 mm
• Facemask	+2 to +4 mm

Occlusal Plane

For each distal movement of point A, it will drop down by $\frac{1}{2}$ mm. Superimpose mark 2 on the old menton and facial plane, parallel the mandibular planes by rotating at menton, now construct the occlusal plane.

Dentition

Lower Incisor: Lower incisor position is determined by arch length. The following are considered requirements when lower incisors are drawn:

- Occlusal plane relation.
- A Pg line relation.
- Symphyseal relation.

To mark the position of lower incisor, a dot is placed (relating to lower incisor tip) so that it is 1 mm above the occlusal plane and 1 mm ahead of A Pg line. Superimposition is on corpus axis. Draw the lower incisor in ideal relation to A Pg line and occlusal plane.

Lower Molar: Without treatment, the lower molar will erupt directly towards the occlusal plane. With treatment, the lower molar might move forward. Every 1 mm forward of the lower molar arch length will decrease by 2 mm. Movement of lower molar is decided depending on the lower incisor position.

Upper Molar: Upper molar is drawn to class I relation with the lower molar.

Upper Incisor: Upper incisor is drawn on good overjet and overbite (2.5 mm) relation to lower incisor. Interincisal angle of 130° is maintained.

Soft Tissue

- **Bridge of the nose:** Superimpose nasion along facial plane and palatal plane. The prediction is moved back 1 mm/year along the palatal plane. Tip of the nose is now traced and merged with the superior aspect of the nose drawn.

- *Upper lip:* The difference in position between the old and new upper incisors is the correction. This distance is divided into three parts and marked 1, 2, 3. Superimpose along the facial plane and registering at its intersection with the occlusal plane, trace the upper lip in the following manner:
 - Soft tissue thickness of the upper lip does not change, so by superimposing old and new point A, soft tissue point A is traced.
 - Upper lip tracing is done by superimposing the tip of upper incisor on mark 1 (parallel to the occlusal plane).
- *Lower lip:* Bisect the overjet and overbite in the original tracing and mark points. Superimposing on the interincisal points keeping the occlusal plane parallel, trace the lower lip and soft tissue point B. Lower lip thickness does not change as well. Lip strain is eliminated if there has been any in the original tracing. Soft tissue point B is traced.

Disadvantages of Roentgenographic cephalometric (RCM) methods (Moss, Salentijn and Skalak):

- Using radiographic cephalometry growth can be observed in two dimensions only.
- Growth behavior can be observed only relative to an arbitrarily chosen point location and reference line direction.
- Growth behavior of an individual differs greatly when studied by different radiographic methods.
- Cephalic regions with little or no growth may appear to rotate or translate with respect to some arbitrarily fixed reference points.
- It is impossible to construct a model of growth using a cephalogram that is independent of the choices of fixed point and fixed line.
- The methodological constraints using RCM include (i) the anatomical points used are relatively widely spaced relative to the dimensions of skull (ii) with RCM, only the motion of the selected points can be analyzed and depicted and (iii) in reality, some of the intervening points may not be actively growing and RCM is incapable of discriminating these points.
- RCM methods are incapable of correctly depicting in local detail time related changes of biologic shape or of changes in location of biologic shapes; only anecdotal observations are possible.

Morphometrics Methods

Morphometrics is defined as the mathematical or graphic description of shape and of shape change in time, including changes of both size and location as well as changes in location alone. The various morphometrics methods of growth prediction includes: biorthogonal analysis introduced by Bookstein, allometric centered models, and finite element method (FEM).

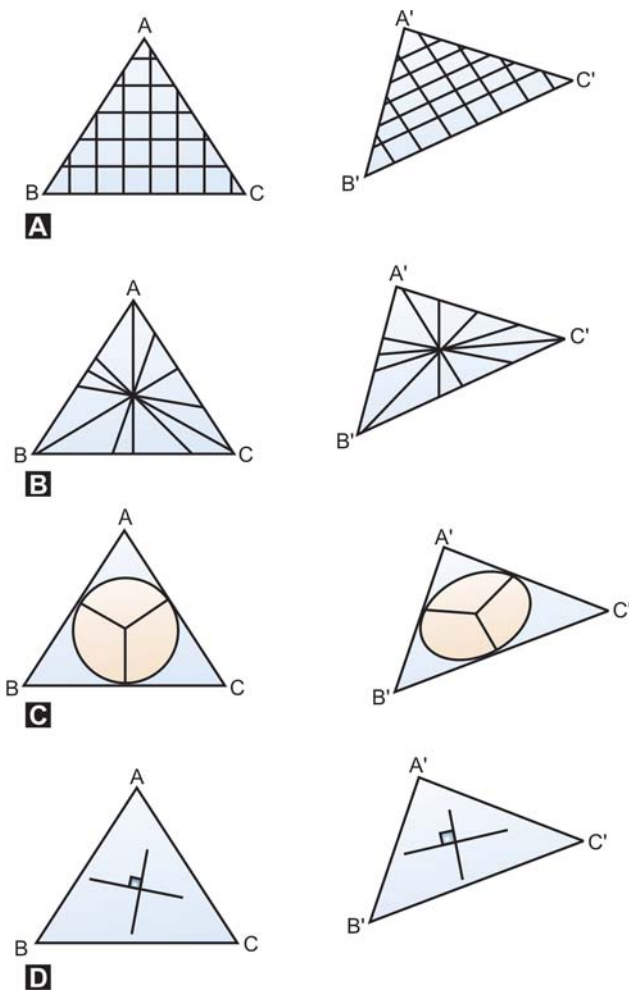
Biorthogonal Analysis

Bookstein Source describes some geometric manipulations by which mean growth invariants can always be extracted from observed configurations of landmarks. Biorthogonal analysis is the expression of a rigorous new approach to growth prediction. Biorthogonal analysis prescribes a mutual orientation for the two triangles for which the principal axes are aligned (Figs 12.32A to D).

The four kinds of invariants: The simple geometry of this construction leads quickly to four basic classes of invariants which can be identified in any smooth deformation.

Ratio of sides: Figure 12.33 indicates how directions placed symmetrically with respect to the principal axes are dilatated at the same rate by the shape change. Then the ratio of their lengths is unchanged over the transformation. In particular, if either principal axis of a deformation is parallel to the bisector of one of the triangle's vertex angles, then the ratio of the sides which meet at that vertex will be unchanged.

Angle: Angles with the axis of compression, or straddling it, as in Figure 12.34A, must open; those involving or straddling the axis of extension must shrink. Those which span both the axes or neither, will increase or decrease as the expanding or shrinking sector dominates. A bit of elementary trigonometry, omitted here, indicates that those angles remain constant for which $\tan \theta_1 \tan \theta_2 = d_2/d_1$, ratio of the two dilatations in Figure 12.34B. For small changes, where d_2/d_1 is very close to unity, these correspond to angles with $\theta_1 + \theta_2 = 90^\circ$ —lines symmetrically placed about the bisectors of the principal axes. Whenever a bisector of the principal cross is aligned with the bisector of one of the triangle's angles (Fig. 12.34C), that angle will remain approximately constant over the transformation.



Figs 12.32A to D: The method of biorthogonal directions for two triangles. A: The uniform transformation of interiors they suggest. B: Dilatations of lengths in various directions. C: Dilatations may be represented by the radii of the ellipse into which a circle is deformed. D: The principal directions are axes of this ellipse, and the principal dilatations are proportional to their lengths

Proportional division: If one principal axis is parallel to a side of the original triangle, then the third vertex may be viewed as displaced directly away from the foot of its perpendicular with respect to that side. Hence, whatever the dilatations, the proportion in which the foot of its perpendicular divides that edge is unchanged over the deformation.

Ratio of perpendiculars: If each of the principal axes makes an angle of 45 degrees with one side of the starting

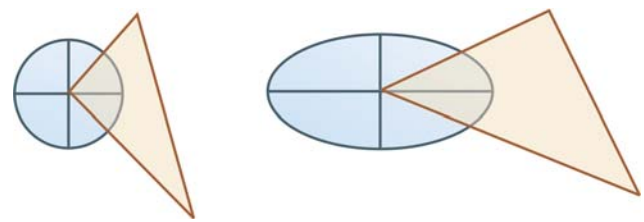


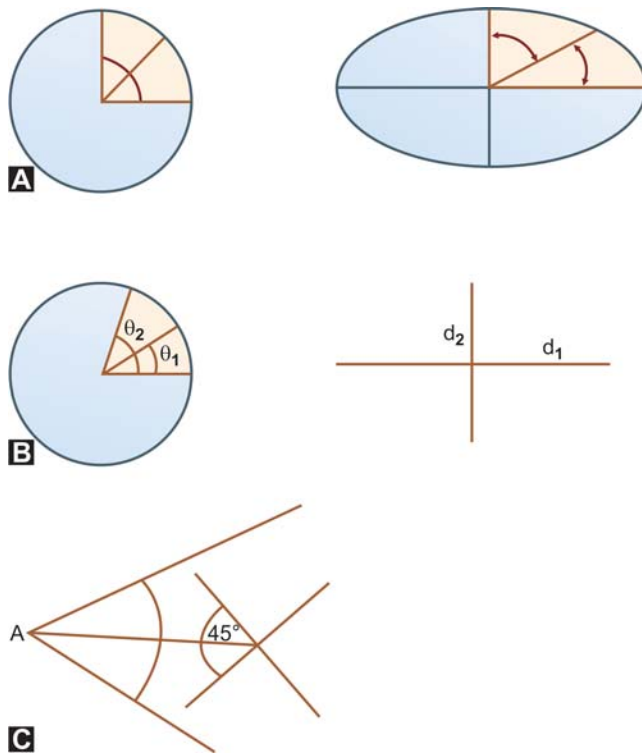
Fig. 12.33: Invariant ratios. Segments at equal angles to the principal axes of a deformation are dilatated at the same rate

triangle, then both that side and the altitude to it from the third vertex are dilatated at the same rate (as their directions are symmetric with respect to the axes). Hence, as shown in Figure 12.33, the aspect ratio of this triangle, its height divided by its base, will be approximately invariant over the deformation.

Generality of these constructs: It is not necessary that any of the sides or angle bisectors of the triangle of landmarks lie along the principal axes or their bisectors for there to be invariants. We may define homologous segments through any points of the triangles, not only those which happen to be named landmarks. In Figure 12.34A, for instance, the lines at 45 degrees to the principal axes are parallel to transects connecting vertex A to points 0.2 and 0.8 of the distance from vertex B to vertex C, segments whose ratio is therefore unchanged by the deformation. In Figure 12.34B, the principal axes lie along lines from A to a point 0.7 of the way from B to C and from B to a point 0.8 of the way from A to C, so the angle between these lines is exactly unchanged at 90 degrees over the deformation. A slowly changing length-ratio corresponds to a rapidly changing angle, and conversely. In Fig. 12.13,A, as the dotted lines are dilatated at the same rate, the angle between them has altered a great deal. In Figure 12.34, B, because the dashed lines are at an invariant angle of 90 degrees, the proportion between them has altered more than any other shape measure. It thereby, becomes the best scale for discriminating before from after, diagnosing anomalies, and characterizing treatment effects.

Allometric Model of Growth

Analysis of craniofacial skeletal growth are materially assisted by development of appropriate models. Mathematically, such models should closely describe and



Figs 12.34A to C: Invariant angles. A: Angles straddling the axis of compression open; those straddling the axis of extension close. B: Those angles are constant for which $\tan \theta_1 \tan \theta_2 = d_2/d_1$. C: For small changes in shape these are angles bisected by the bisectors of the principal axes

graphically illustrate the kinematic behavior of the growing skeleton. The advantages of allometric model are as follows:

- The allometric network model permits the display of the kinematic behavior occurring when there are marked transformations of particular cranial skeletal regions during growth.
- The allometric network model permits growth prediction.
- The growth network model has a significantly smaller error than a centered model when modeled and the actual biologic data are compared.

Allometric Networks

In growing biologic systems, allometry expresses the concept that a constant ratio exists between the specific growth rates of a pair of quantifiable variables during

some time interval. This relationship, as originally expressed by Huxley, is

$$y = b \times k$$

where k represents the constant ratio between the specific growth rates of x and y that grow allometrically.

The allometric centered model was one way by which some degree of allometry could exist in a growing craniofacial skeletal system. Another way to achieve some degree of allometry is to assume that there exists a network of points in a plane (two-dimensional) or in space (three-dimensional), so that the length of each line element between points has its own specific growth rate. For example, in Figure 12.35, l_1 , l_2 , and l_3 are each assumed to have differing specific growth rates. These three line elements cannot all grow allometrically if θ is fixed; however, if the line elements are allowed to rotate, then each line element may grow allometrically. The future geometry of the triangle at any time is determined (“predictable”) if the initial size and shape are given and the growth rates of the three sides are known.

When networks more complex than a triangle are considered, the main problem is to determine the conditions under which allometric growth is possible, and the main answer is that allometric growth is possible, with arbitrary specific growth rates for each line element, if the network forms, what is known in structural analysis as a statically determinate truss. For this purpose, the definition of simple networks is similar to the definition of simple trusses. A truss is a network in which the line segments are solid bars, as in a bridge truss. In the

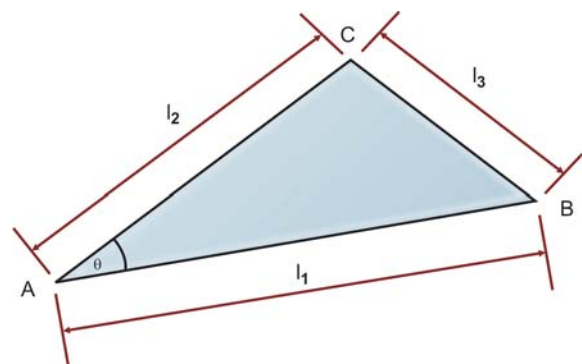


Fig. 12.35: The three line elements (l_1 , l_2 , l_3) of this figure can all grow allometrically with different growth rates only if it is not fixed and the line elements are allowed to rotate. If all growth rates are equal, the angles are constant, but not otherwise

allometric networks considered here, the line segments are not considered to be material bars and may, in fact, consist of different material points at different times.

A plane network will be called simple if it can be drawn in the following way: Start with a triangle made up of three segments and three nodes. Then add one node at a time, connecting it to two previous nodes by two new segments. If the entire network can be generated in this way, it is called a simple plane network.

A space network will be called simple if it can be drawn in the following way: Start with a tetrahedron made up of four nodes and six segments that form the edges of the four triangular faces of the tetrahedron. Then, add one node at a time, connecting it to three previous nodes by three new segments. If the entire network can be generated in this way, it is called a simple space network.

The allometric network model has been shown to be superior to the allometric centered model in the analysis of craniofacial skeletal kinematic growth behavior in three ways: (1) The allometric network model permits growth prediction, when allometric constants are known; (2) The network model has significantly smaller errors than the centered model; and (3) The network model is capable of displaying growth kinematics of both the neural and facial skulls while marked transformations such as relative rotation of two sets of cranial anatomic points occur in time. It is important to note that the allometric network model is not an alternative method of roentgenographic cephalometry; for example, the segments of maximal superimposition in representations of best fit between actual and theoretical rat and human cranial growth data indicate only those regions with respect to which growth is most nearly allometric. The predictability of the allometric network model, while real, is related to these same superimpositions of best data fit and should not be understood as referring to any possibly underlying biologically dynamic process(es). Despite the evident advantages of the allometric network model relative to an allometric centered model, the network model does not exhibit all possible attributes of cranial skeletal growth kinematics. For example, the network model displays differing shapes, in time, but not shapes change, per se, in the sense of Bookstein. The third of the methods, that of a finite element model, is better able to provide a detailed description of cranial skeletal growth change.

Finite Element Method (FEM)

The conceptual bases of the mathematical modeling of craniofacial skeletal growth presented must be carefully described since they introduce terms, concepts, and procedures that are not commonly used by students studying growth. In a technical sense, two separate ideas are presented. The first is that the use of a growth strain tensor permits a fundamental description of growth; more specifically, that a *growth tensor* is a fundamental descriptor of growth. The second idea is that the use of the finite element method makes possible a convenient and systematic approximation of the mean growth tensor within finite elements, using the data from a finite number of data points. It will be helpful to define and exemplify both concepts, first that of finite elements and then, that of growth tensors.

Finite elements: The fundamental attribute of finite element is its ability to subdivide structures or bodies into large number of two or three dimensional elements each of which is called as "*finite element*". Each of the finite elements can be analyzed individually as well as together with other elements. The growing head for example is considered as a continuum that is subdivided into two-dimensional study by a series of imaginary lines or by plane surfaces in a three-dimensional study. The point of connection of each line with another line is called "*nodes*". In the three-dimensional case, the planes are bounded by straight lines that extend from one node to another. These lines and planes conceptually subdivide the body into a series of contiguous finite elements, a process termed *discretization*. The location of the node connecting two or more such lines is occupied by a single point whose coordinates are assumed to be known. Figure 12.36 illustrates a three-noded, two-dimensional finite element (a triangle); an eight noded, three-dimensional hexahedron (a box); and a four-noded tetrahedron (a pyramid); there are many other possible configurations.

Finite elements may also be constructed with curved lines or surfaces as boundaries. Such elements require one or more additional nodes between the corner nodes described above. Such elements are described in the references to the FEM cited above and may be advantageous if sufficient nodal data are available so that

element boundaries can fit curved anatomic shapes closely. Only straight-line and plane boundaries are used to illustrate the principal attributes of the FEM more simply. Practically, discretization may be carried out by either arbitrary selection of some of the particles of the body as nodes or in the case of skull, anatomical points can be used. These anatomical points may be identical with the points of regular points used in RCM like N, S, and ANS. The FEM differs significantly from RCM in the method of analysis of growth behavior. Whereas in RCM only the behavior of a set of separate points (for example, Na, Ba) is studied, in the FEM the emphasis is on analysis of growth within each element. In the method presented here, the assumption is made that all the points within a given finite element share a common growth behavior. This will become more accurate as the number of elements is increased and their size is decreased. Let us consider a two-dimensional, three-noded, triangular finite element (Fig. 12.36). At some initial time (t_0) the coordinates of each of the three nodes are assumed to be determined and, in time, as a result of growth, the location of these nodes is transformed, so that, at some later time (t_1) the new coordinates of the three nodal points are again determined. In the common clinical vocabulary, the triangular finite element is said to have “grown larger,” while in the more precise vocabulary of continuum mechanics, it is said that the finite element has been deformed, strained, or transformed. The term strain may be understood in the sense that it is used in classical physics or mechanics. When a physical body is externally loaded, it tends to deform. For example, in vertical, axial, compressive loading, a physical body tends to shorten vertically and widen horizontally. The dimensional changes, expressed as fractions of the original lengths, are strains, and can be measured.

The force per unit area required to produce such strain is termed stress and is a measure of the internal resistance of the body to deformation. In growth, the deformations are not due to stresses but are produced by cell division, cell growth, and production of extracellular matrices (that is, by adding mass). Accordingly, a growth strain is the measurable deformation of a biologic body resulting from its growth. With the use of the mathematical definitions introduced by Skalak and associates and the numerical methods detailed by Patel, a quantitative description of the values of the growth strains, or deformations or

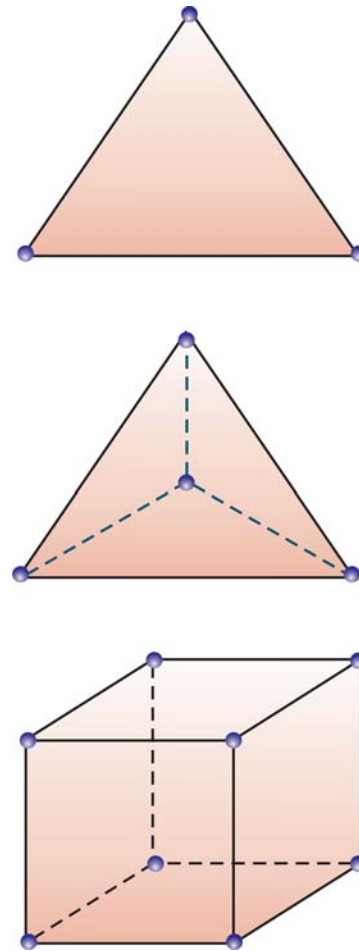


Fig. 12.36: Three possible finite elements: a three-noded, two-dimensional element (a triangle); a four-noded element (a pyramid); and an eight-noded element (a box)

extensions, as well as a determination of the principal directions of these extensions, can be computed and graphically displayed. It is assumed that the growth description of any single element is valid for all of the points within the continuum of that same element in the present study of skulls.

Analysis of Growth Behavior

FEM provides a description of the growth behavior of the entire continuum of points within each finite element. The growth behavior described includes both alterations in the distances between points in the continuum and in their spatial relationships. Let us consider the two-dimensional triangular finite elements

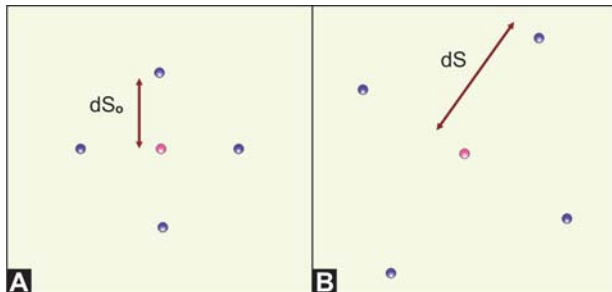
described in figure 12.37. The area of this element is considered to be filled completely by a very large number of points and together all these points constitute a continuum. At some original state or time, each of these points is considered to be equally separated from all other points by some identical very small, yet finite distance in the two dimensional plane formed by this element. In the original state (S_0), the distance in the original state is designated as dS_0 . In response to the biological process of growth, the finite element gets strained and therefore the area also increases dS_0 . As shown in the diagram (Figs 12.37A and B), the elongation has taken place along the long axis of principal direction of growth or in other words, due to growth, finite element has been strained. This has produced maximal elongation of the continuum of points in one direction and minimal elongation of continuum of points in perpendicular direction. Thus, FEM permits quantitative descriptions of maximal and minimal growth strains and of the relative position of the principal axes of these growth strains for each age. Accordingly, FEM analysis gains in accuracy as the number of finite elements studied is increased and their size decreased.

Growth Tensors

While customary roentgenographic cephalometry (RCM) usually employs vectors to display and describe results, the FEM computes growth tensors. The difference between these two descriptions is related to the different basic viewpoints of the two descriptors of growth. Conventional RCM reports the results in terms of displacements or movement of points in time. The continuum viewpoint suggests that the fundamental effects

in growth are the local extensions (growth strains) in the vicinity of any point. These growth strains are distributed throughout the growing body and are properly described by the growth tensor at each point. This calls for a tensor description of growth extensions as opposed to a vector description of displacement, as given by RCM. In a technical sense, the tensor description of growth is derived from displacements via the theory of continuum mechanics. More precisely, the growth tensor is a function of the derivatives of displacements with respect to position (that is, the displacement gradient). The tensor description provides an invariant (coordinate system independent) description of growth at each point. With the example of the deformation of a single three-noded, triangular finite element given above, it is possible to provide a two-dimensional elucidation of the term tensor as used here. This is a concept that is not in general use in physical anthropology or in orthodontics, but is common in continuum mechanics.

Tensors, in general, are abstract mathematical entities that cannot be readily visualized. This lack of visualizability is the greatest stumbling block in comparison to the type of graphic representation customary with vectors. The general concept of a tensor involves a set of numbers that describe a three-dimensional field, such as a stress or strain (or a mathematical operation, such as transformation or coordinates). The numbers that specify a tensor are called components. A tensor may also be specified by giving so-called principal directions and principal values that may be represented graphically. Generally, the tensor is a descriptor. The numbers and figures are representations of the tensor or the expression of the tensor in a particular coordinate system. As a tensor is a descriptor, so a growth tensor is a descriptor of growth. It may be regarded as specifying a transformation of coordinates from one stage of growth to another. A simple analogy of a tensor operation may be helpful. Arithmetic addition is a mathematical operation. The concepts of arithmetic addition, subtraction, etc. per se, are without specific (dimensional or directional) content, and this is true also for the concept of a tensor operation. The addition of 3 and 5 to produce the sum of 8 is an arithmetic operation. The integers involved do not express the concept of addition, but only the input and output of its operation. Addition, per se, is a concept in which the results of the operation are easily observed. A tensor is a more general concept than arithmetic but has rules analogous to arithmetic addition, multiplication, etc. In



Figs 12.37A and B: Effect of strain on continuum of points in a finite element. In initial state, S_0 is considered as separate from neighboring points by equal distances dS_0 . In final state due to growth, there is an increase in the distance dS in only one direction

the present context, tensor operations are involved with the transformations from one set of coordinates to another set, and the results may be expressed as the growth strain tensor.

The growth tensor describes the relative displacements of all points in the neighborhood of the given point (for example, displacements of the continuum of points within a given finite element relative to any selected point) (Fig. 12.38). It will be remembered that a vector quantity (a force, an acceleration) has two attributes of magnitude and direction. In three dimensions, a growth tensor has attributes of six scalar components or three principal directions. In the present context, a growth tensor may be regarded as specifying the local transformation from one set of coordinates to another (from the initial to the final coordinates in the interval of growth considered). For example, it describes the transformation of a triangular finite element at time t_0 in a Cartesian frame of reference to the location of the same triangular element after growth at time t_1 . Strictly speaking, the growth tensor allows determination of displacements only within a rigid body translation and/or rotation. This freedom is one aspect of the fact that the growth tensor is independent of any

rigid body motions or displacements of the subject of study on radiographs representing it.

The analysis of a transformation by the finite element method requires the ability to locate each node before and after the transformation and to determine accurately the three-dimensional coordinates of each node both before and after the transformation. Technically, this is the ability to “map” homologous points from one time frame to another. It may also be stated as the ability to identify end points of the path of a particular point in space-time. It is precisely these relationships between homologous points that a growth tensor describes and quantifies. In principle, if the growth tensor is given at every point of a body (and it may vary continuously from point to point) it gives sufficient information to reconstruct final form (size and shape) in three dimensions, starting from the initial form; again, this is within an arbitrary rigid body motion. The difficulty with RCM is that it specifies the particular rigid body translation and rotation to be used and thus lacks independence of these choices.

There are, in general, various types of tensors of geometric interest: a metric tensor, a rotation tensor, a rate-of-growth tensor, etc. An additional example may be useful. Consider a human skull, in *norma verticalis*, in which the sagittal suture is marked by implants at points bregma and lambda and the location of the coronal and lambdoid sutures is marked by implants at pterion and asterion, respectively. In the customary three-dimensional Euclidean space, all three sutures and the implants have a fixed vectorial (spatial) relationship to each other. Now, allow this skull to be rotated relative to the fixed three-dimensional reference frame, so that the lateral surface of the skull is now in view. With respect to the fixed axes of the reference frame, the coordinates of all the implanted points of that skull have changed; however, within the skull, the relative relationships of these points and of the vectors of the sutures to each other have remained constant. Such constancy of description of growth in the face of rigid body rotation is expressed by the growth tensor. This statement reiterates one of the chief advantages of the finite element method over RCM: that tensor descriptions are independent of external frames of reference. It is this attribute that permits the tensor descriptions of the FEM to overcome the principal geometric disadvantage of all RCM and to permit the FEM to be independent of any method of registration and orientation.

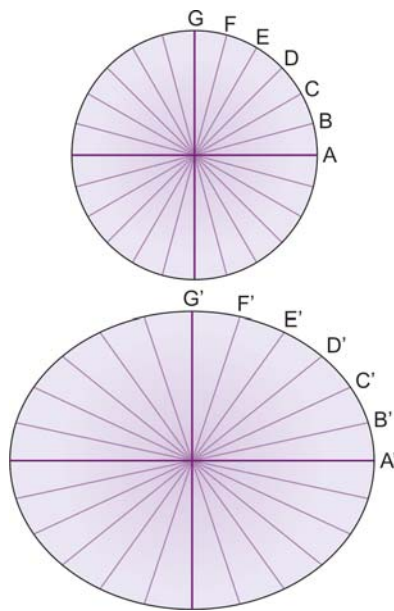


Fig. 12.38: The deformation of the circle above to the ellipse below illustrates the general transformation of the neighborhood of any point during a two-dimensional growth. The lines through the point are shown in their corresponding initial and final positions. The general line segment both stretches and rotates, but the principal axes are subject to stretching only

A further advantage is that growth is described in a local basis. In the vicinity of each point, the local changes due to growth are described independently of that which is taking place elsewhere. In RCM the displacements of any point may depend on the growth at distant points.

Advantages of FEM

- The principal advantage of the finite element method is that it can provide an estimate of the growth behavior of all points of the body and in all directions at all points.
- FEM offers significant analytical advantages over RCM. These are related to the demonstration that the FEM descriptions are invariant and independent of any local reference frame; that is, the descriptions and comparisons are independent of the well-known difference existing between many of the usual methods of superimposition and registration of different RCM analytical methods.
- Any point used can be reliably identified and that the coordinates of its location can be determined in a two-dimensional plane, at least, or in three dimensions if possible.
- Considering the methodological constraints detailed for the RCM methods, it is clear that, with the use of any RCM, we can describe and depict only the relative growth behavior of a series of discrete anatomic or material points (that is, relative to the selected fixed point or arbitrary frame of reference); nothing is known of the growth behavior at the individual points in the continuum between the discrete points actually studied.

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Growth Rotations

CHAPTER OUTLINE

- Implant Radiography
- Mandibular Growth Rotations
- Bjork's Classification
- Bjork and Skeiller's Method
- Schudy's Concept
- Dibbets' Concept
- Proffit's Classification
- Growth Rotation of Maxilla
- Jaw Rotation and Tooth Eruption
- Mutual Relationship of Rotating Jaw Bases
- Studies on Growth Rotations
- Cephalometric Diagnosis in Growth Rotation

The phrase *growth rotation* was introduced in 1955 by Bjork, who used it to describe a particular phenomenon occurring during the growth of the head. Ever since the introduction of the term rotation, it has been attended by a considerable amount of confusion. The many and often, conflicting interpretations of rotation advanced in the literature make it evident that the different contexts of usage of the term have created real problems.

The technique, whereby metal implants are inserted in bone has been used in animals for more than a century, but the application of the method in craniometric studies of growth in man is of a more recent date. This paved the way for studying the growth of mandible in terms of rotation. Professor Bjork is considered the father of Implant Radiography. Cephalometric implant radiography has revolutionized the growth studies in the field of orthodontics.

The direction of growth and inclination of jaw bases is unique and no two individuals are alike. Of all the patterns of growth, growth rotations assume an important

role in orthodontics because of its major impact on treatment strategies. The rotation of maxillary and mandibular jaw bases is a major factor in etiological assessment, determining the nature of anomaly, the prognostic evaluation, determining the possible forms of treatment, in choosing the principles of treatment and also in assessing the stability of treatment results. Certain rotational patterns of jaw bases can be manipulated quite effectively by means of functional and orthopedic devices while certain extreme rotations are very difficult to treat and surgical correction has to be performed at a later stage.

IMPLANT RADIOGRAPHY

In implant radiography, metallic implants have been inserted in the jaws to serve as fixed reference points. The first implant radiographic study was initiated in the year 1951 by Bjork and comprised of a mixed longitudinal study of about 100 children of each sex covering the age period from 4 to 24 years. The sample consisted of normal children with and without malocclusion and also children with pathologic conditions. By means of the implant method, it is possible to locate sites of growth and resorption in the individual jaws and to examine individual variations in direction and intensity. The marker technique has also proved useful in the analysis of the mechanisms underlying changes in the intermaxillary relationship during growth, an analysis that has led to a radical modification of previous views. This applies in particular to the vertical jaw relation, since the implant technique detects considerably greater rotation of the mandible during growth than that may be observed with conventional methods.

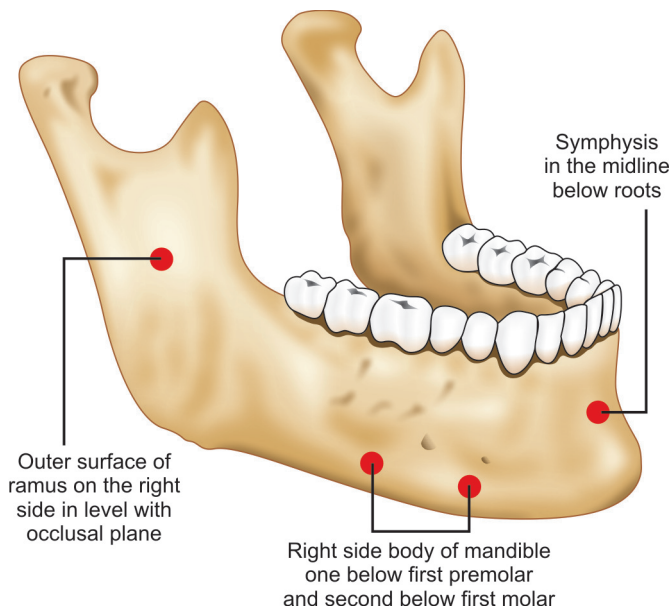


Fig. 13.1: Sites of implants in mandible

Technique of Implant Radiography

In this procedure, inert metal pins are placed in the mandible. Tantalum inert pins which are 1.5 mm long and have 0.5 mm diameter are used. These metal pins get fused to the bone (Fig. 13.1). Osseointegrated implants serve as reference points and serial cephalometric radiographs are taken repeatedly over a period of time and compared. Rotation of jaw bones was estimated using implant radiography only. The different sites of implant placement are given in Table 13.1.

Information obtained from implant radiography is as follows:

Site of growth	: Very accurate information
Amount of growth	: Very accurate information
Rate of growth	: Relatively accurate
Direction of growth	: Very accurate
Type of study	: Longitudinal study
Draw backs	: Two-dimensional study of three-dimensional process and radiation hazard.

MANDIBULAR GROWTH ROTATIONS

Mandibular rotations assume an important role in orthodontic treatment planning because mandibular

Table 13.1: Site of implants

Bone	Site
Mandible	1. Symphysis in the midline below roots. 2. Right side body of mandible—one below first premolar and second below first molar. 3. Outer surface of ramus on the right side in level with occlusal plane.
Hard palate	1. Behind canines. 2. Front of first molar in the junction between alveolar process and palate.
Maxilla	1. Inferior to anterior nasal spine. 2. Bilaterally in the zygomatic process.

rotations are more common than maxillary rotations. Mandibular inclination drastically affects facial morphology, and treatment planning and treatment outcome and mandibular inclination to a certain extent can be effectively guided by certain functional or orthopedic appliances during growth period. Implants were placed in the following sites of mandible:

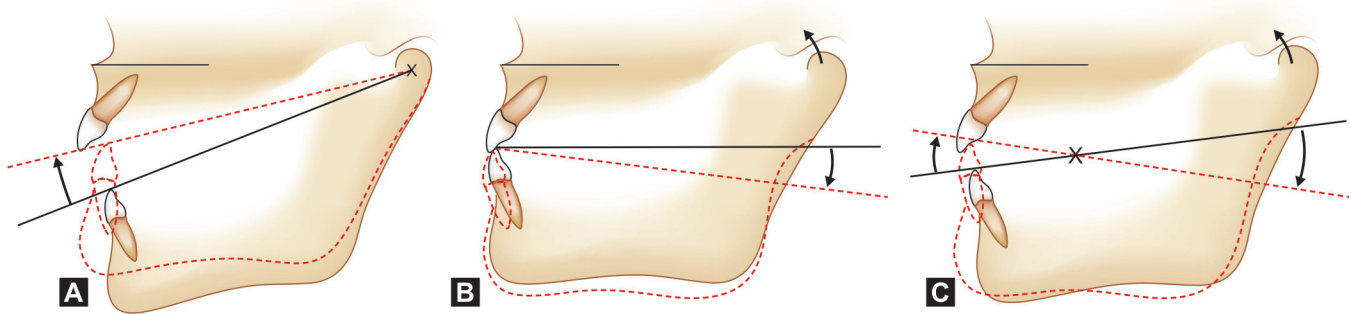
- Anterior aspect of symphysis, in the midline below the root tips.
- Two pins on the right side of the mandibular body. One pin under the first premolar and the other below the second premolar or the first molar.
- One pin on the external aspect of the right ramus in level with the occlusal surface of the molars.

By superimposing two consecutive tracings of child's mandible registered on implants, Bjork found that the image of the older mandible had appeared to have rotated slightly forward during the intervening period.

BJORK'S (1969) CLASSIFICATION

Bjork has classified rotation of mandible into forward and backward rotations. *Forward rotation* has three types and occurs in the following ways (Figs 13.2A to C):

Type I. In this type (the one that is usually considered) there is forward rotation about centers in the joints which gives rise to a deep-bite, in which the lower dental arch is pressed into the upper, resulting in underdevelopment of the anterior face height. The cause may be occlusal imbalance due to loss of teeth or powerful muscular pressure. This lowering of the bite may occur at any age.



Figs 13.2A to C: Forward rotation of the mandible with the center (Type I) at the joints (A), with the center (Type II) at the incisal edges of the lower incisors (B), and with the center (Type III) at the premolars (C)- Bjork (1969)

Type II. Forward growth rotation of the mandible about a center located at the incisal edges of the lower anterior teeth is due to the combination of marked development of the posterior face height and normal increase in the anterior height. The posterior part of the mandible then rotates away from the maxilla. The increase in the posterior face height has two components. The first is the lowering of the middle cranial fossa in relation to the anterior one as the cranial base bends, and the condylar fossa then being lowered. The second component is the increase in the height of the ramus, which is pronounced in case of vertical growth at the mandibular condyles. Only the latter component, which is the larger one, is illustrated in Figure 13.2, and is described as follows. Because of the vertical direction of condylar growth, the mandible is lowered more than it is carried forward. The lowering of mandible takes place as a forward rotation in relation to the maxilla because of the muscular and ligamentous attachments, with the center at the incisal edges of lower incisors. The eruption of the molars keeps pace with the rotation. Due to the simultaneous marked resorption below the gonial angle, the height in this region may not increase to a great extent and the lower border undergoes a characteristic remodeling.

Type III. In anomalous occlusion of the anterior teeth, the forward rotation of the mandible with growth changes its character. In case of large maxillary overjet or mandibular overjet, the center of rotation no longer lies at the incisors but is displaced backward in the dental arch, to the level of the premolars. In this type of rotation, the anterior face height becomes underdeveloped when

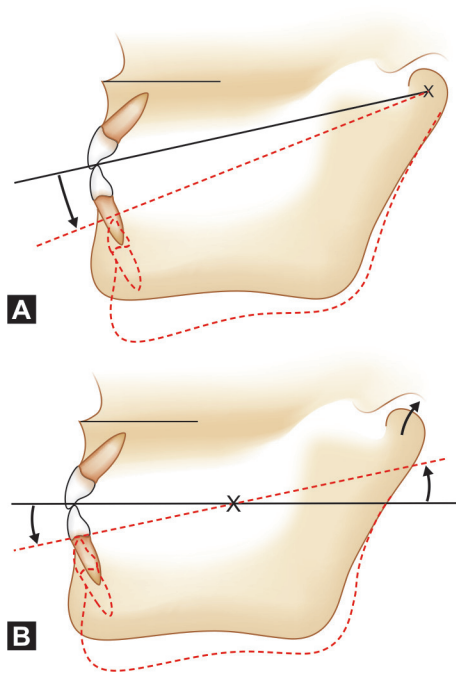
the posterior face height increases. The dental arches are pressed into each other and basal deep-bite develops.

In the growth rotation of types II and III, the mandibular symphysis swings forward to a marked degree and the chin becomes prominent. This is one of the reasons for the chin formation characteristic of man. The inclination of the teeth is also greatly influenced by the rotation of the jaw. The position of the lower incisors seems to be functionally related to the upper incisors, as is reflected by the fact that the interincisal angle undergoes a smaller change than the rotation of the jaw. As a result, the incisors in their eruption are guided forward and there is an increase in the alveolar prognathism right down to the apical zone. This is contrary to the impression given by the jaw profile. However the rotation, also displaces the paths of eruption of all the teeth in the mesial direction, thereby tending to create crowding in the anterior segment through what may be referred to as “packing”.

The rotation also affects the position of the lower posterior teeth in relation to the upper teeth. Forward growth rotation thus causes the lower posterior teeth to be more upright than usual in relation to the upper posterior teeth, with an increase in what may be called interpremolar and intermolar angles.

Backward rotation of the mandible is less frequent than forward rotation and has been examined by the implant method in considerably fewer subjects. Two types have been recognized (Figs 13.3A and B):

Type I: Here, the center of the backward rotation lies in the temporomandibular joints. This is the case when



Figs 13.3A and B: Backward rotation of the mandible with the center at the joints (A) and with the center at the last occluding molars (B)

the bite is raised by orthodontic means, by a change in the intercuspation or by a bite-raising appliance, and results in an increase in the anterior face height. Backward rotation of the mandible about a center in the joints also occurs in connection with the growth of the cranial base. In the case of flattening of the cranial base, the middle cranial fossae are raised in relation to the anterior one, and then the mandible is also raised. There may be other causes also, such as an incomplete development in the height of the middle cranial fossae, as in oxycephaly. This underdevelopment of the posterior face height leads to a backward rotation of the mandible, with overdevelopment of the anterior face height and possibly open-bite as a consequence. The mandible is, in principle, normal.

Type II. Backward rotation occurs about a center situated at the most distal occluding molars. This occurs in connection with growth in the sagittal direction at the mandibular condyles. As the mandible grows in the direction of its length, it is carried forward more than it is lowered in the face, and because of its attachment to muscles and ligaments it is rotated backward. Because

of the position of the center of rotation at the molars, the symphysis is swung backward and the chin is drawn back below the face. The soft tissues of the chin may not follow this movement, and a characteristic double chin can form. Basal open-bite may develop, and there is difficulty in closing the lips without tension. Since the position of the lower incisors, as mentioned earlier, is functionally related to the upper incisors, they become retroclined in the mandible and the alveolar prognathism is reduced. The lateral teeth are not guided distally in their eruption to the same extent, and crowding tends to develop in the anterior segment of the lower arch.

Because of the backward rotation of the mandible, the interpremolar and intermolar angles are small, which means that the premolars and molars are inclined forward in relation to the maxillary ones, and usually to a pronounced degree, because of the close proximity of these teeth to the center of rotation.

This type of backward rotation has been found to be characteristic in cases of various forms of condylar hypoplasia also.

There is an obvious relationship between the type of rotation of the mandible and the direction of condylar growth. Bjork states that the explanation for this remains to be found, but it is evident that muscular factors play an important part.

Structural Signs of Growth Rotation

From the clinical standpoint, it is important to detect extreme types of mandibular rotation occurring during growth. Bjork has given seven structural signs of extreme growth rotation in relation to the condylar growth direction. Not all of them will be found in a particular individual, but the greater the number which is present, the more reliable the prediction will be (Fig. 13.4). Moreover, it is evident that these signs are not so clearly developed before puberty. The seven signs are related to the following features: (1) Inclination of the condylar head; (2) Curvature of the mandibular canal; (3) Shape of the lower border of the mandible; (4) Inclination of the symphysis; (5) Interincisal angle; (6) Interpremolar or intermolar angles; and (7) Anterior lower face height.

BJORK AND SKEILLER'S METHOD

Bjork and Skeiller subsequently together carried out extensive implant studies and introduced various

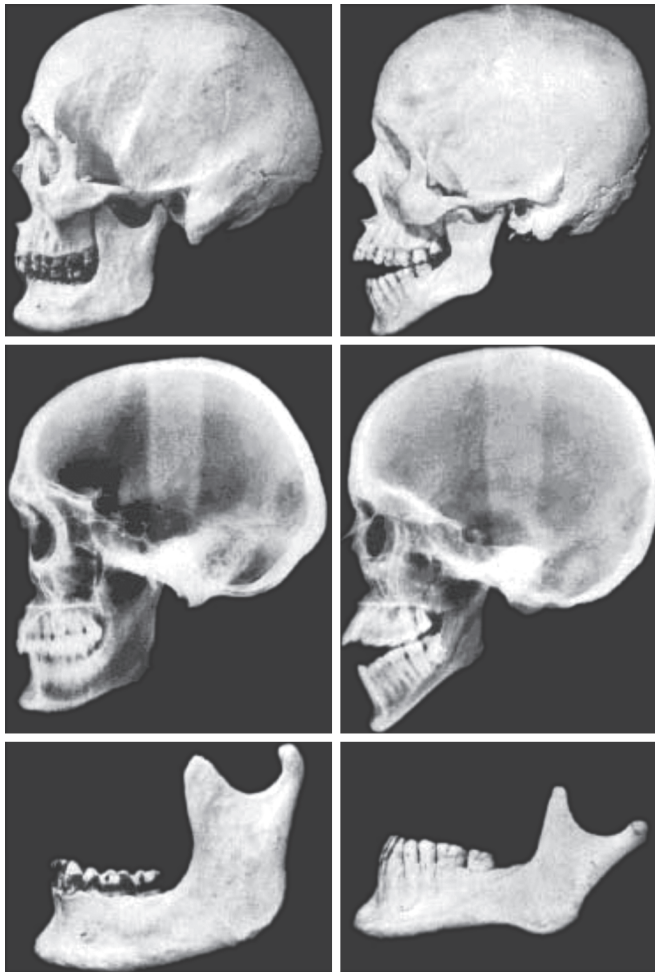
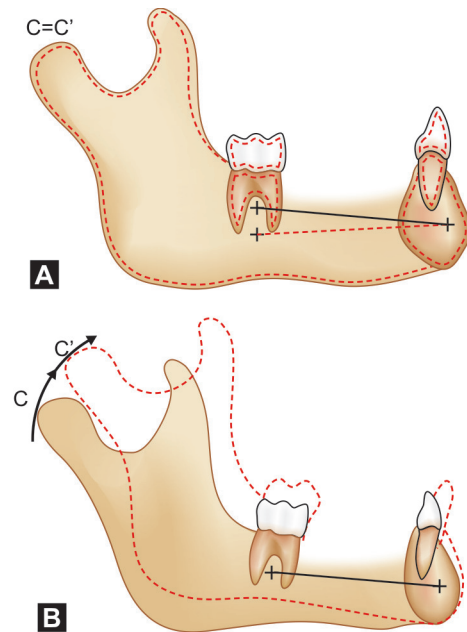


Fig. 13.4: Structural signs of mandibular growth rotation demonstrated in two craniums—one with basal deep-bite and one with basal open-bite. (Bjork 1969)

terminologies to understand the rotational pattern of mandible. They divided the rotation into three components:

- **Total rotation.** The rotation of the mandibular corpus measured as a change in inclination of an implant line in the mandibular corpus relative to the anterior cranial base.
- **Matrix rotation.** This is the rotation of the soft-tissue matrix of the mandible relative to the cranial base. The soft-tissue matrix is defined by the tangential mandibular line. The matrix rotation has its center at the condyles.
- **Intramatrix rotation.** The difference between the total rotation and the matrix rotation is an expression of

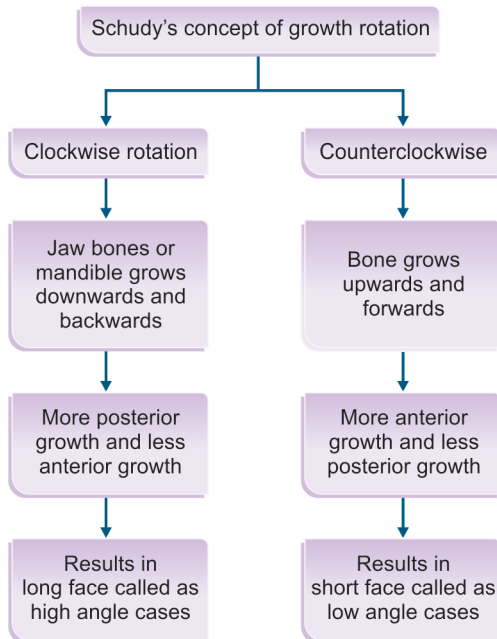


Figs 13.5A and B: Schematic illustration of intramatrix rotation: (A) Two mandibles superimposed on their external contours. Note divergence of the implant lines indicative of intramatrix rotation not reflected in dimensional change or alteration of mandibular contours. (B) The same two mandibles superimposed on the implant markers. Note lack of concordance of mandibular contours, indicating extensive remodeling during development

the remodeling at the lower border of the mandible. It is identified by the change in inclination of an implant or reference line in the mandibular corpus relative to the tangential mandibular line. The intramatrix rotation has its center somewhere in the corpus (Figs 13.5A and B).

SCHUDY'S CONCEPT (Flow chart 13.1)

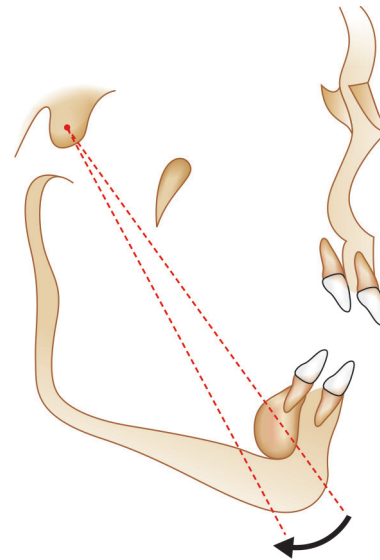
According to Schudy, the rotation of the mandible is the result of disharmony between vertical growth and anteroposterior or horizontal growth of jaws. Accordingly, he describes two type of growth rotation of mandible, namely, clockwise and counter clockwise rotations. If the condylar growth is greater than vertical growth in the molar area, the mandible rotates counterclockwise and results in more horizontal change of the chin and less increase in anterior facial height. Extremes of this condition causes closed bites. Conversely, if vertical

Flow chart 13.1: Schudy's concept of growth rotation

growth in the molar region is greater than that at the condyles, the mandible rotates clockwise resulting in more anterior facial height and less horizontal change of the chin. Extremes of this condition cause open bites. Schudy states that the rotation of mandible has more implications in vertical growth of the craniofacial complex.

The Clockwise Rotation (Fig. 13.6)

Clockwise rotation of the mandible is a result of more posterior vertical growth than condylar growth, the point of rotation being the condyles. If vertical growth in the molar region is greater than that at the condyles, the mandible rotates clockwise resulting in more anterior facial height and less horizontal change of the chin. Extremes of this condition cause open bites. Growth at the mandibular condyles produces a forward component of the chin, not a downward and forward component. When the vertical increments of facial growth begin to assert their influence on condylar growth through occlusal contact, then a downward and forward direction of the chin is produced. Thus, it can be said that condylar growth is pitted against the combined vertical elements of growth. The final vector of growth of the chin is a resultant of the struggle between horizontal growth and

**Fig. 13.6:** Clockwise rotation of mandible

vertical growth, in other words, between condylar growth and vertical elements. These vertical components are:

- Growth at the nasion and in the corpus of the maxilla which produces an increase in the distance from nasion to anterior nasal spine and causes the maxillary molars and posterior nasal spine to move away from the sella-nasion plane;
- Growth of the maxillary posterior alveolar processes causing the molar teeth to move away from the palatal plane; and
- Growth at the mandibular posterior alveolar processes causing the molar teeth to move occlusally.

The vertical growth of the anterior alveolar processes does not seem to have an appreciable effect on facial height. It is merely expressed in varying degrees of overbite.

The dorsal migration of the glenoid fossa is a very real factor in many cases and tends to cancel out the growth of the condyles; thus, in a sense, it is arrayed on the side of vertical growth.

When vertical growth exceeds horizontal growth (condylar growth), pogonion cannot keep pace with the forward growth of the upper face and the mandibular plane must become steeper. This has an influence on the treatment also. Obviously, this condition would not help reduce the ANB angle, and it would not aid in correction of a class II molar relation.

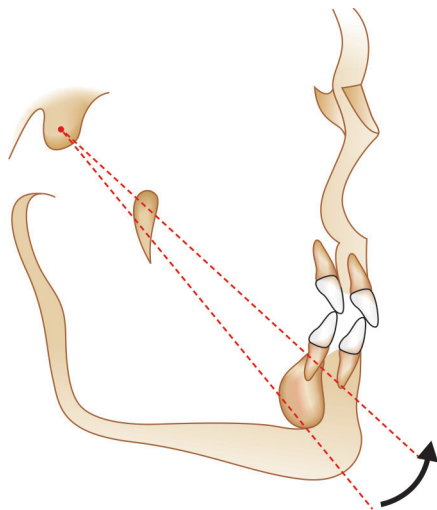


Fig. 13.7: Counterclockwise rotation of mandible

Counterclockwise Rotation (Fig. 13.7)

Counterclockwise rotation of the mandible is a result of more condylar growth than combined vertical growth. This type of rotation is nearly always accompanied by a forward movement of pogonion and an increase in the facial angle. The point of rotation is the most distal mandibular molar in occlusal contact. This "flattening" of the mandibular plane tends to increase the vertical overbite and renders vertical overbite correction and retention more difficult.

The size of the gonion angle has an important influence upon the number of degrees of resultant counterclockwise rotation. The smaller the gonion angle, the greater the rotation which is produced for each millimeter of forward movement of pogonion. When this angle is extremely small, it results in extreme flattening of the mandibular angle together with forward growth of the pogonion.

Schudy states that the mandible should not be considered as a single growth entity, but rather as four entities: (1) Growth of the condyle and ramus; (2) Growth of the corpus; (3) Growth of the posterior alveolar process; and (4) Growth of the anterior alveolar process (Figs 13.8A to E).

This unique bone grows in many different ways. It may grow quite uniformly in all directions or in any one of its aspects which may grow out of proportion to the rest of the bone. The condyles may grow rapidly while

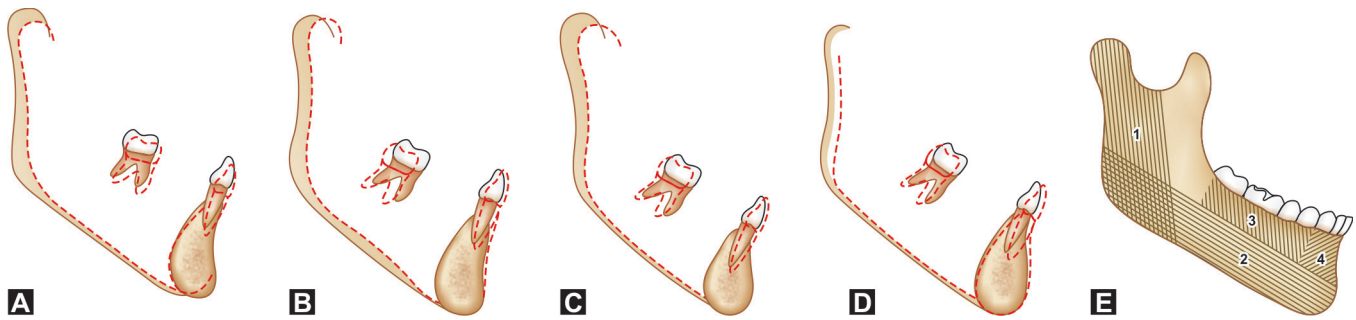
the corpus grows very little or none. The corpus may increase considerably in length while the condyles may exhibit little or no growth. The vertical growth of the anterior alveolar process may exceed that of the posterior process. The converse may also be true. The condyles may grow out of proportion to the posterior alveolar processes and vice versa. All of these patterns have an effect upon vertical overbite and overjet.

DIBBETS' CONCEPT

Dibbets re-examined the concept of "intramatrix rotation" as defined by Bjork and Skeiller in 1983. Bjork and Skeiller postulated that "intramatrix rotation" is an expression of the remodeling at the lower border of the mandible and assumed that the rotation occurred in the corpus of the mandible. Lavergne and Gasson, on the other hand, contended that the rotation affected the ramus and the gonial angle and, consequently, the length of the condylion-pogonion diagonal. An alternative interpretation of mandibular rotation was presented by Dibbets in order to overcome the controversies of intra-matrix rotation.

Dibbets hypothetically constructed two possible divergent patterns of mandibular growth: (1) a *circular growth pattern* which postulates condylar growth as a segment of a circle with its center at the chin. The whole mandible would then rotate around itself within its periosteal contours, resulting in "intramatrix rotation" without enlargement of the mandible. (2) The other pattern is conceived as a *linear growth pattern* of the condyle, without any "intramatrix rotation," and maximum enlargement of the mandible. Most children will be observed to fall in between these two postulated extreme patterns.

Comparing the extremes, Dibbets deduced that "intramatrix rotation" is capable of offsetting growth and of neutralizing growth to a substantial degree. It does so by inducing a curvilinear growth direction for the mandibular condyle, more than is necessary to account for mandibular enlargement. The process involves a selective remodeling of the mandible. Thus, Dibbets introduced the concept of *counterbalancing rotation* which tries to offset condylar growth increments, which would otherwise throw the mandible out of its established equilibrium with its associated skeletal units (that is, the maxillae). Thus, counterbalancing rotation is a



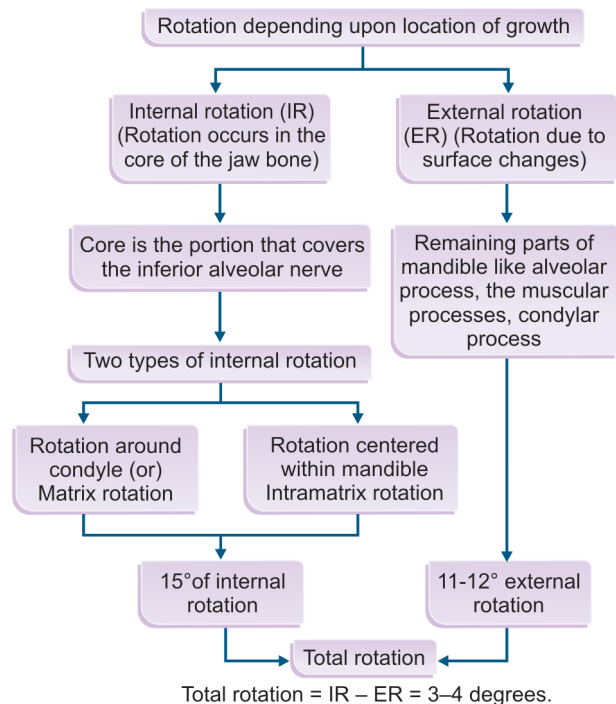
Figs 13.8A to E: Different types of mandibular growth. (A) Much condylar growth but very little growth of corpus. (B) Good corpus growth but almost no condylar growth. (C) Molar grew vertically much more than incisor. (D) Incisor grew more than molar. (E) Arbitrary segments of mandible: (1) Growth of the condyle and ramus; (2) Growth of the corpus; (3) Growth of the posterior alveolar process; and (4) Growth of the anterior alveolar process

mechanism that (1) neutralizes growth and (2) results in selective enlargement of the mandible.

PROFFIT'S DESCRIPTION OF ROTATION (Flow chart 13.2)

Proffit coined different terminologies to explain growth rotation of mandible, namely total rotation, internal rotation and external rotation. *Total rotation* is the net resultant rotation including the internal and external rotation. *Internal rotation* is the rotation that occurs in the core of the jaw. In the mandible, the core is the bone that surrounds the inferior alveolar nerve (Fig. 13.9). The internal rotation is masked by surface changes and alterations in the rate of eruption of teeth. There are two contributions to internal rotation, namely matrix and intramatrix rotations. *Matrix rotation* occurs around the condyle while *intramatrix rotation* is centered within the body of mandible (Figs 13.10A and B). Matrix rotation is also termed as hinge rotation. This is the rotation of the mandibular plane related to cranial base. This is quantified as angular changes in the mandibular plane relative to S-N or Frankfurt horizontal. Because the center of rotation is around the condyle, one can visualize how the opening or closing of the bite will change the inclination of mandibular plane relative to S-N. Intramatrix rotation is the rotation of bony element within its periosteal matrix which occurs in the corpus or the core of the mandible. If there is no hinge movement, then the total rotation and intramatrix rotation will be identical. When the mandibular plane opens or closes, then the total rotation is modified even though the

Flow chart 13.2: Proffit's description of rotation



intrinsic remodeling of the mandible (intramatrix rotation) is presumably not affected. On an average there is about 15 degrees of total or summary rotation from age 4 to adult life. Of this, about 25 percent results from matrix rotation and 75 percent from intramatrix rotation.

External rotation is the result of surface changes. These surface changes include resorption in the posterior part

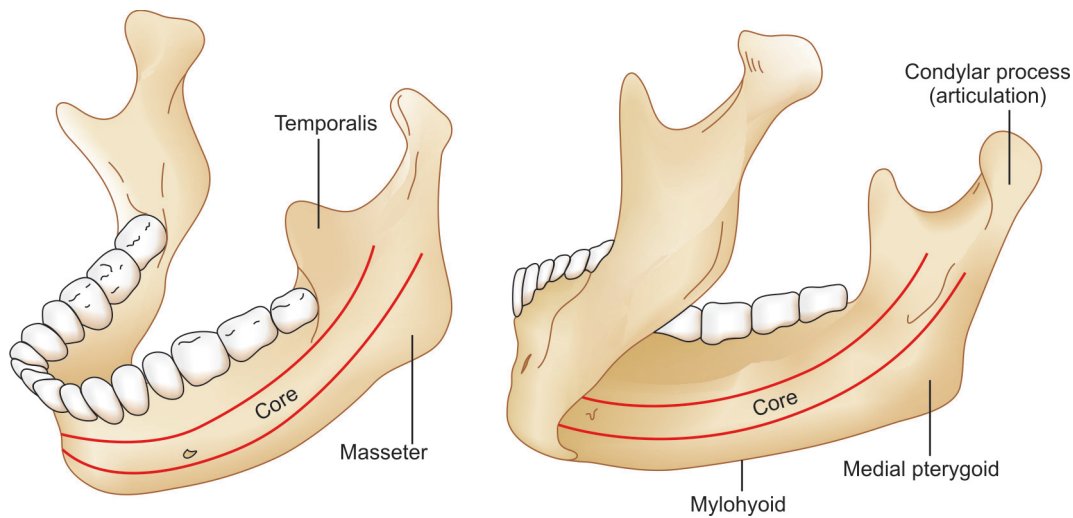
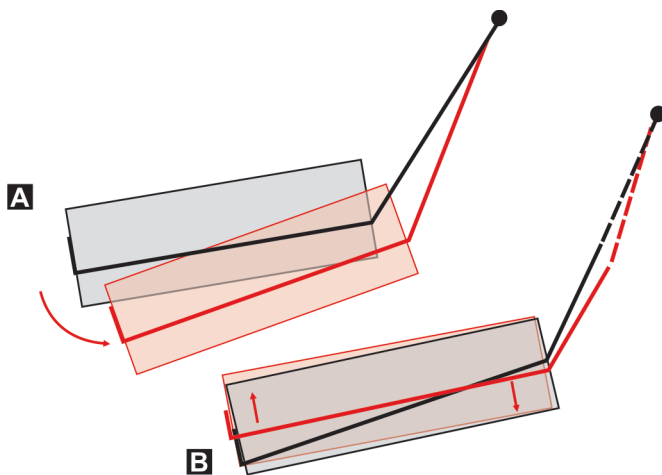


Fig. 13.9: Core and functional processes of mandible



Figs 13.10A and B: Components of internal rotation. (A) Matrix rotation or rotation centered on the condyle and (B) Intramatrix rotation or rotation centered within the body of mandible

of the lower border of the mandible, while the anterior aspect of the lower border is unchanged or undergoes slight apposition. This external compensation in an average growing adult is about 11 to 12 degrees. The orientation of the jaw results from a combination of both internal and external rotation. The difference between the internal and external rotation accounts for 3 to 4 degrees reduction in mandibular plane angle during growth in adolescence.

In short face type, there is excessive forward rotation of mandible due to increase in internal rotation and decrease in external rotation. A high angle case shows backward rotation due to lack of normal internal rotation. Sometimes, the high angle cases show backward external rotation.

GROWTH ROTATION OF MAXILLA

Maxilla undergoes extensive remodeling and displacements when subjected to various functional demands. Generally, the vector of maxillary growth is in anterior and inferior direction (downward and forward displacement). The growth of nasomaxillary complex is due to two basic methods namely passive displacement and active growth. Thus, both passive displacement and active growth of nasomaxillary complex carries it downward and forward. This anterior and inferior thrust is not always uniform.

Due to the varying growth activities of middle cranial fossa, the sutural attachments of midface and surface remodeling, the maxilla tends to get rotated by displacement. Sometimes, this rotational pattern is extreme which could result in canting and misfit of the palate and maxillary arch.

Bjork and Skeiller (1972) studied this rotational growth of maxilla with the help of implants. Those sites that were selected for implant placement by Bjork include:

- Inferior to anterior nasal spine.
- In the zygomatic process of maxilla (lateral implant).
- At the border between the hard palate and the alveolar process medial to first molar.

Later on, several methods of maxillary superimposition were described in the literature. Various sites in maxilla were selected, some of which include ANS, PNS, pterygomaxillary fissure and internal palatal structures. It was concluded that various structures within the maxilla like the palatal plane, ANS, PNS, A point, exhibited varying degrees of rotation and the entire maxilla also demonstrated varying degrees of rotation in relation to cranial base. So superimposition with implants based on these structures was not always reliable.

The lateral implant placed on anterior and posterior contour of zygomatic process seems to give best results when compared to other sites. Serial superimposition with the lateral cephalograms at these implant sites revealed varying degrees of rotation of jaw bases. Based on these studies, Bjork and Skeiller introduced various terminologies to describe the growth rotation of maxilla. They include:

Internal rotation: This is the rotational pattern that occurs in the core of the maxilla. This is also called intramatrix rotation. The internal rotation is similar to intramatrix rotation of mandible.

External rotation: Simultaneous to internal rotation of maxilla, varying degrees of resorption of bone on the nasal side and apposition of bone on the palatal side in anterior and posterior parts of the palate also takes place. Similarly, variations in the amount of eruption of the incisors and molars occur. All these changes collectively contribute to external rotation. This external rotation is usually opposite in direction and equal in magnitude to the internal rotation, so that the two rotations cancel each other and the net change in jaw orientation, as evaluated by the palatal plane is zero. However sometimes, variations from the average norms do occur and extreme rotational patterns can occur during growth. Depending upon the different degrees of combination of internal and external rotations, Bjork and Skeiller observed two types of rotational growth. The terminologies they used are forward and backward rotations.

Forward growth rotation: This condition occurs either due to excessive internal rotation or lack of normal

compensatory external rotation or a combination of both.

Here, the maxilla is inclined upward and forward, that is, the anterior end is tipped up. This is also called ante inclination as coined by Schwarz. He also named this condition as pseudo-protrusion. This actually aggravates maxillary protrusion. This forward rotation also tends to tip the incisors forward, increasing their prominence (Fig. 13.11). The extent of forward tipping in relation to anterior cranial base is given in degrees by Schwarz. The inclination angle is not measured directly but is defined as the angle between the Pn-perpendicular and the palatal plane (J angle). In ante inclination, this angle is greater than 85 degrees whereas in normal inclination, this angle is approximately equal to 85 degrees.

Backward rotation: Backward rotation of maxilla is exactly opposite to that of forward rotation where there is downward and backward tipping of the anterior end of the palatal plane and the maxillary base. This is otherwise called as retroinclination, a term coined by Schwarz. In this type of maxillary displacement, the jaw bases are translated posteriorly and the upper incisors appear to tip lingually. The angle between palatal plane and the anterior cranial base is lesser than the normal value. Here, the angle between Pn-perpendicular and palatal plane is less than 85 degrees.

Though the growth related rotation does occur in the midface, extreme rotational patterns are often compounded with various environmental disturbances. According to Linder-Aronson, Lowe, and Woodside, various environmental influences such as neuromuscular

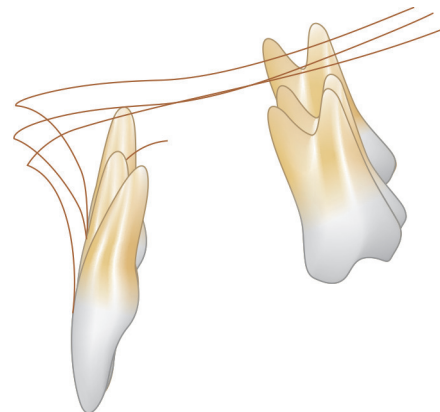


Fig. 13.11: Internal rotation of maxilla

dysfunction, occlusal forces, gravity and nasorespiratory malfunctions can cause extreme rotations of jaw bases. An upward and forward tipping of the anterior part of maxilla is often confirmed in mouth breathers, while a downward and backward tipping of anterior part of maxilla is observed as a natural compensation in patients with vertically growing faces. The inclination of maxilla can be influenced by both fixed mechanotherapy and functional, orthopedic treatment measures which have to be carefully monitored during active treatment. It has been established that the degree of maxillary rotation shows variations in direction and in intensity each year. It is smaller, in absolute value, than the degree of mandibular rotation. Due to the fact that the direction of the maxillary and mandibular rotations is not always the same, it appears that the interaction between the maxillary and mandibular rotations plays an important role in the vertical and sagittal relationships of both jaws.

JAW ROTATION AND TOOTH ERUPTION

By means of the implant method, the eruption paths of the teeth have been analyzed by Bjork and Skeiller in relation to facial development and growth of the jaws; growth changes were followed in a longitudinal series of profile radiographs. The rotation of the face necessitates compensatory adaptation of the paths of eruption of the teeth. When there is full compensatory occlusal development, the lower incisors retain their inclination in the face practically undisturbed, irrespective of the rotation of the jaw, because of a forward tipping on the jaw base. The posterior teeth in the lower jaw, too, are involved in this compensatory occlusal development and are, likewise, tipped forward. The lower dental arch then shifts forward on the jaw base without undergoing any appreciable change in shape. The intermolar inclination remains comparatively constant as the lateral teeth in both jaws follow the rotation of the face. Eruption of the upper molars appeared to be a combination of active eruption of the teeth in the jaw bone and bodily rotation of the maxilla. In forward rotation of maxilla, the incisors tend to tip forward increasing their prominence, while in the backward type of rotation, the anterior teeth are directed more posteriorly, relatively decreasing their prominence and uprighing them. The eruption path of mandibular teeth is normally upward and forward. In excessive

forward rotation in short faced individuals, the incisors tend to be carried into an overlapping position even if they erupt slightly, hence there is a tendency towards deep bite malocclusion. Due to the lingual movement of lower incisors, there is reduction in arch length because of rotational changes and this is more evident the in mandible when compared to the maxilla.

MUTUAL RELATIONSHIP OF ROTATING JAW BASES

When Bjork introduced the concept of rotation to orthodontics after using metallic implants, the concept was widely extended and misused. In an attempt to clarify this situation, a classification was proposed whereby a clear-cut distinction between the morphogenetic and positional rotations was presented. Morphogenetic rotation of the mandible concerns the shape of the mandible itself, while positional rotation deals with the position of the mandible. Maxillary and mandibular rotations were demonstrated to have a major role in the mutual adjustment of both the jaws (Fig. 13.12).

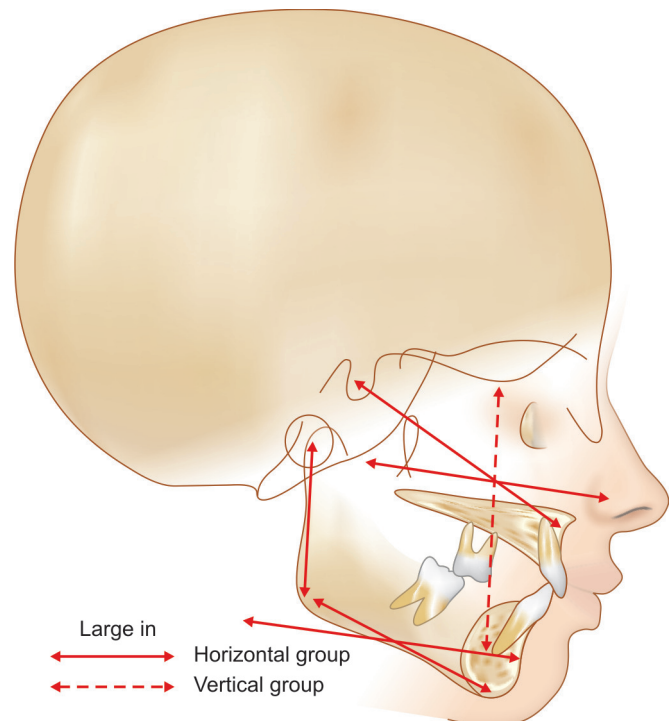


Fig. 13.12: Facial areas that were larger in the horizontal or vertical group patients. Arrows demonstrate dimensions that were larger in the horizontal group and area that was smaller in the horizontal group and larger in the vertical group

Four types of mutual rotation of jaw bases were proposed by Laverne and Gasson after extensive implant studies (1982). This is important clinically because dentoalveolar malocclusion depends on the combination of these rotations.

- *Convergent rotation of jaw bases:* This rotation results with closing of maxillo-mandibular plane angles creating a severe true deep bite that is difficult to manage. Both maxilla and mandible converge towards each other (Fig. 13.13).
- *Divergent rotation of jaw bases:* In this type, the maxilla and mandible move away or diverge from each other (Fig. 13.14). This rotation leads to the opening of the basal angle and can result in open

bite problems. Extreme cases of divergent rotation requires surgical correction.

- *Cranial rotation of maxilla and mandible:* In this type, both the maxilla and mandible rotates upward and forward. This horizontal growth pattern occurs in a relatively harmonious manner wherein rotation of maxilla occurs upward and forward and compensates for cranially rotating mandible, offsetting a deep bite. This compensation results in normal overbite relationship (Fig. 13.15).
- *Caudal rotation of maxilla and mandible:* In this type, both the maxilla and mandible rotate downward and backward. Similar to cranial rotation of jaw bases, this also occurs in a harmonious manner wherein the downward and backward maxillary rotation offsets the open bite created by downward and backward mandibular rotation (Fig. 13.16).

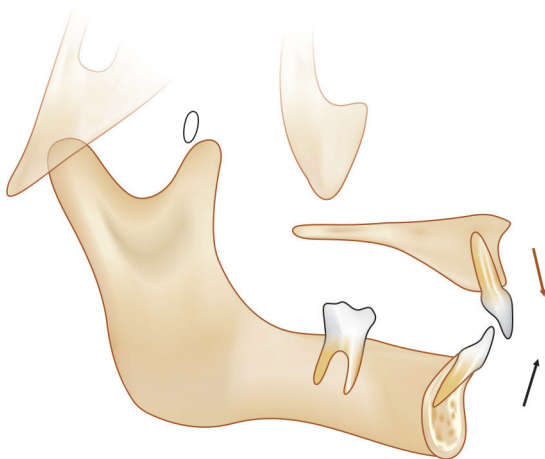


Fig. 13.13: Convergent rotation of both maxilla and mandible

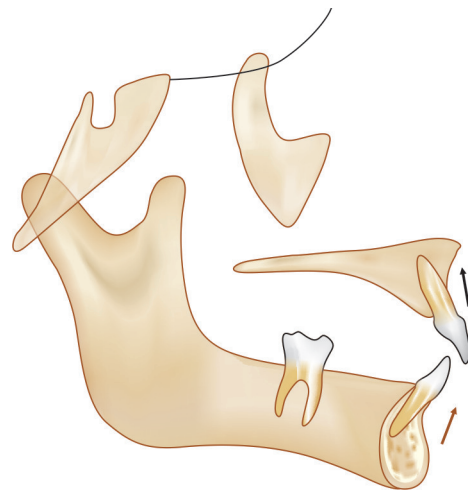


Fig. 13.15: Cranial rotation of maxilla and mandible

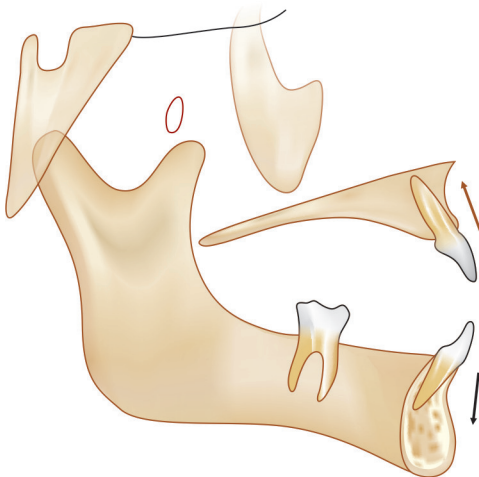


Fig. 13.14: Divergent rotation of maxilla and mandible

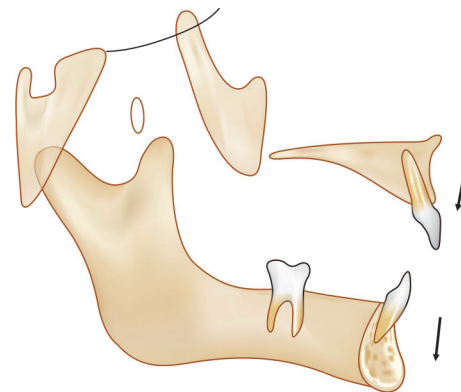


Fig. 13.16: Caudal rotation of maxilla and mandible

STUDIES ON GROWTH ROTATIONS

Numerous authors have contributed to the understanding of growth rotations through implant studies. Notable among them are Bjork, Skeiller, Laverne and Gasson, Schudy, Proffit. Various methods and studies have been described to predict the direction and the amount of growth rotation of mandible. Some of the recent classical studies have been described here.

Skeiller, Arne Bjork and Linde-Hansen Method (1984)

This is a longitudinal cephalometric study with the aid of implants. They chose 21 Danish children both girls and boys who never had orthodontic treatment. Implants were placed at specific sites in the mandible and were followed for a period of six years. Totally, two cephalometric X-rays were taken. The first radiograph was taken three years before the pubertal growth spurt and second, three years after the pubertal growth spurt. Changes in the inclination of the implant in relation to NSL have been designated as negative when the implant line rotated forward and as positive when the implant line rotated backward.

Initially, they chose 44 independent morphologic variables in the mandible for superimposition. From them, 4 variables were used, which in combination gave the best prognostic estimate of the mandibular growth rotation (Fig. 13.17).

- Mandibular inclination represented by three alternatives:
 - Index I—proportion between posterior and anterior facial height
 - Lower gonial angle (GOL)
 - Inclination of the lower border (NSL-ML1)
- Intermolar angle (MOLs-MOLi)
- Shape of the lower border (ML1-ML2)
- Inclination of the symphysis (CTL-NSL)

Index I was found to be more accurate than the other two and was chosen as the first independent variable. This is then co-related with the 2nd, 3rd, and the 4th variable. It was found that combination of these variables gave the best prognostic estimate (86%) of mandibular growth rotation. Besides these variables, the other morphological signs mentioned in Table 13.2 can also be used for growth rotations.

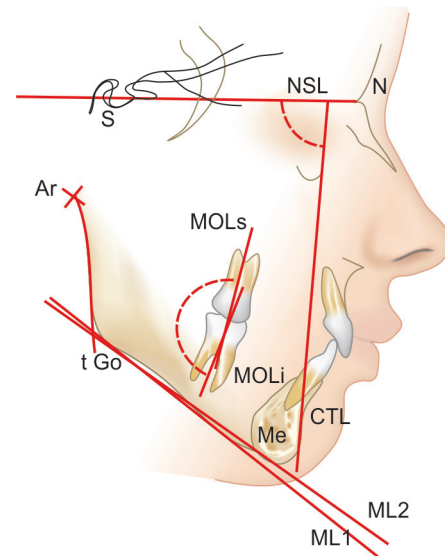


Fig. 13.17: Variables used by Skeiller, Arne Bjork and Linde-Hansen

Method by Todd, Ram S Nanda, Frans Currier, Surendar K Nanda

The purpose of this study was to determine whether symphysis morphology could be used as a predictor of direction of mandibular growth rotation. They used lateral X-rays of 115 adults for this study.

The mandibular symphyseal dimensions studied included symphyseal height, symphyseal depth,

Table 13.2: Morphological observations seen in forward and backward rotations by Skeiller, Arne Bjork and Linde-Hansen

No.	Parameter	In forward rotator	In backward rotator
1.	Anterior facial height	Decreased	Increased
2.	Inclination of lower border of mandible	Curves downward	Notched
3.	Intermolar angle	Vertical or obtuse	Acute
4.	Inclination of symphysis	Slopes backward	Slopes forward
5.	Interincisal angle	Vertical or obtuse	Acute
6.	Inclination of condyle	Curves forward	Straight or slopes back
7.	Curvature of mandibular canal	Curved	Straight

symphyseal angle and symphysis ratio—Height/Depth ratio (Figs 13.18A and B).

A tangent is drawn passing through point B. Then a grid was constructed with the lines of the grid parallel and perpendicular to the constructed tangent line. The superior limit of the symphysis was taken at point B, and the most inferior, superior, anterior, and posterior outline of the symphysis are marked and grid was thus completed. The symphyseal height was defined as the distance from the superior to the inferior limit of the grid. The symphyseal depth is defined as the distance from the anterior to posterior limit on the grid. The symphyseal angle is formed between the lines passing through point B and menton and the mandibular plane. The 115 adults are separated groups based on gender and the four symphyseal measurements are then divided

into small, average and large groups. The direction of mandibular growth for the above individuals was evaluated with seven cephalometric measurements that included Y axis, SN to mandibular plane, palatal to mandibular plane, gonial angle, sum of the saddle, articulare and gonial angles, percentage of lower facial height, and posterior to anterior facial height.

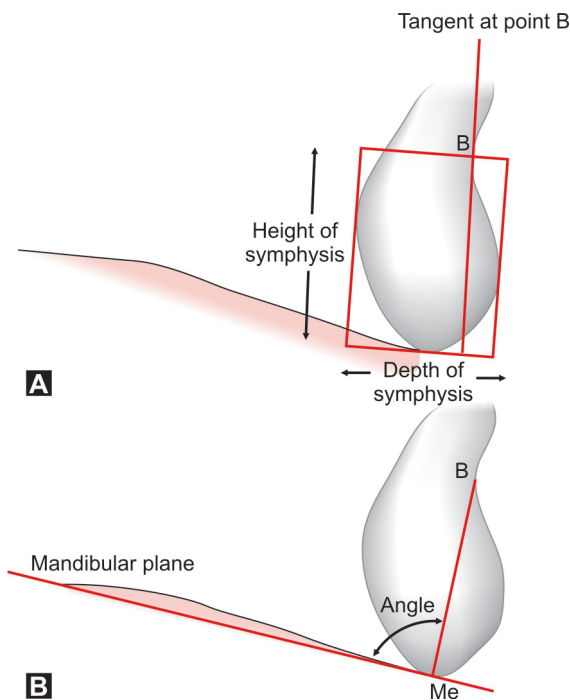
Inferences

- This study concluded that the mandible with anterior growth rotation was associated with small height, large depth, small ratio, and large angle of the symphysis. Conversely, a posterior growth rotation was associated with a large height, small depth and increased ratio, small angle of the symphysis.
- Men possess a stronger relationship between symphyseal morphology and the direction of mandibular growth when compared to women.
- Symphysis ratio was strongly related to the direction of mandibular growth in men.
- Women also showed the same relationship as the men between symphysis height, depth, ratio, and angle to the direction of the mandibular growth.
- Symphysis showed continuous change up to adulthood in both male and female, with the female subjects having smaller and earlier changes occurring than compared to male.
- The symphyseal depth, height, ratio, increased while symphyseal angle decreased with age.

Observations of Petrovic and Stutzman

Various interesting observations were made by Petrovic and Stutzman during their experimental study pertaining to growth rotations. They include:

- A mandibular alveolar bone which was organ cultured for three days showed that the bone turn over adjacent to mandibular first premolar is greater in the anterior growth rotation when compared to posterior growth rotation. Thus, if orthodontic forces are applied, the magnitude of orthodontically induced increase in alveolar bone is statistically high in anteriorly rotating mandible than when compared to a posteriorly rotating mandible.
- This alveolar bone formation is notably greater on the mesial side and resorption is more marked on



Figs 13.18A and B: Cephalometric measurements used to quantify symphysis morphology. (A) Linear measurement. Illustration shows the tangent drawn at point B and parallel and perpendicular lines to this tangent line. The method of measuring height and depth measurements are also shown. (B) Angular measurements. Symphysis angle measured as the posterior-superior angle between lines Me-B point and the mandibular plane. *Source:* AJODO, Volume 1994;60-69

the distal side in the anteriorly rotating mandible. This implies that the first premolar undergoes a physiologic distalization rather than mesialization during growth in the anteriorly rotating mandible. While in posterior growth rotation, the first premolars undergo more mesialization.

- In the anterior growth rotation, the response of prechondroblasts and the subperiosteal and trabecular osteoblasts, muscles of mandible are more responsive to growth stimulating factors like growth hormones—somatomedin, testosterone. This concept is clinically consistent with the finding that thick massive mandible is seen in extreme anterior rotation, gracile ramus and the corpus are seen in extreme posterior rotation.

CEPHALOMETRIC DIAGNOSIS IN GROWTH ROTATION

Several cephalometric measurements have been put forth to identify and predict the rotation and inclination of the jaws. However, it is still very difficult to accurately predict the rotational growth. Baumrind et al concluded that prediction of the direction of mandibular growth rotation by lateral cephalometrics was poor. Similarly, Lundstrom and Woodside have criticized the use of SN base as reference plane to determine the growth rotation of maxilla and mandible, stating that growth direction for each jaw was due to independent factors with only 25 to 40 percent of the variability due to common factors.

Besides these limitations, various cephalometric analysis that are routinely used clinically include:

- Jarabak's cephalometric analysis predicted the direction of mandibular growth from a facial polygon, including the saddle angle (N-S-Ar), articular angle (Ar-Go), and the gonial angle (Ar-Go-Me). With the sums of these three angles greater than 396 degrees, posterior mandibular growth pattern was observed while less than 396 degrees was associated with anterior mandibular growth.
- Ratio of the posterior to anterior facial height of 56-62 percent indicated a posterior growth rotation, whereas a ratio of 65-80 percent indicated an anterior growth tendency.
- If the anterior facial height is greater, the gonial angle adapts to this requirement by increasing. The perpendicular from Go to Sn divides the gonial angle

into a smaller posterior (Go_1) and a larger (Go_2) parts. If gonial angle opens out posteriorly with large Go_1 (posterior rotation), basal plane angle becomes small. If gonial angle opens out anteriorly with small Go_1 (anterior rotation), basal plane angle becomes increased.

- The basal plane angle is the angle between the palatal plane and mandibular plane. It defines the inclination of the mandible to the maxillary base. It determines the direction of rotation of the mandible. Mean angle = 25° . Increased basal angle implies mandible is rotated backwards (vertical growth). Decreased basal angle suggests that mandible is rotated forwards (horizontal growth). The retroclination (-L angle) and anteclination of the maxilla also affects the basal angle. The basal angle is divided into two by the occlusal plane. The upper angle is 110° and the lower is 14° . The lower angle is important for assessing the prognosis for opening the bite. If it is large, the prognosis is good; and if small, the prognosis is poor.
- Besides these, other angles that tell about the rotation of mandible include the Y axis, SN-MP, FH-MP angles.
- Various structural signs of extreme growth rotations can also be elicited from the radiographs. A forwardly rotating mandible shows well defined wider ramus, a relatively straight mandibular canal, wider airway, shorter and broader symphysis, less prominent antegonial notching while backward rotators show a narrow symphysis and ramus, a curved mandibular canal and a prominent antegonial notching.

Growth rotations play a major role in orthodontic treatment planning and outcome. Though various diagnostic methods were evolved to predict growth rotations, none seems to be fulfilling and newer diagnostic methods have to be used in future. Various digitalized cephalometric systems are evolving that will offer more detailed and accurate information. Better therapeutic decisions should be made regarding timing and length of treatment, appliance selection, extraction pattern and possible need for surgery. Therapy should be truly tailored to the individual with the possibility of optimal results in a shorter period of time.

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Chapter 14

Growth Patterns in Skeletal Malocclusion

CHAPTER OUTLINE

- **Growth Pattern in Vertical Skeletal Disproportions**
 - Hyperdivergent growth pattern
 - Hypodivergent facial growth pattern
- **Growth Pattern of Skeletal Class II Malocclusion**
 - Class II division 1 malocclusion
 - Class II division 2 malocclusion
 - Cranial base flexure and class II malocclusions
- **Growth Pattern of Class III Malocclusions**
- **Sexual Dimorphism in Various Malocclusions**
- **Position of Glenoid Fossa in Different Facial Types**

Skeletal malocclusion is a set of human craniofacial morphological characteristics that occurs either due to deficiency or due to an increase in the volume or proportions of the skeletal base. Often skeletal malocclusion is a combination of vertical, transverse and/or anteroposterior discrepancy between the occluding jaw bases, resulting in an improper relationship of the jaws, either in size or spatial positioning. This aberrant growth behavior complicates orthodontic mechanotherapy and treatment planning.

The accurate study of facial skeletal proportions and relationships became feasible with the advent of oriented cephalometric radiography. Since then, various longitudinal and cross sectional cephalometric studies were undertaken to ascertain the growth pattern in various skeletal disproportions.

GROWTH PATTERN IN VERTICAL SKELETAL DISPROPORTIONS

Two commonly encountered extreme facial growth patterns in the vertical plane are “skeletal deep bite” and “skeletal open bite”. Schudy (1964) defined the

former condition “hypodivergent” and the latter as “hyperdivergent” growth pattern.

Schendel described the hyperdivergent growth pattern as “long face syndrome”; Opdebeeck named the hypodivergent pattern as the “short face syndrome.” As the names suggest, the hyperdivergent “long face” is characterized by a tendency toward a relatively large lower face, compared with the hypodivergent “short face”.

According to Schudy, the hyperdivergent and hypodivergent growth patterns have implications not only in the vertical plane of space, but also on the antero-posterior plane. The vertical growth tends to carry the pogonion downward, while anteroposterior growth is attempt to carry it forward. Schudy says this battle ensues early in life and continues till growth is complete and it is the interplay of growth in these two general directions that is responsible for the retrognathic and prognathic facial types.

Bjork and his co-workers coined the term rotations to describe the two types of growth patterns in the vertical plane of space. He coined the term “forward rotation” to describe individuals with short faces and “backward rotation” to describe long face individuals who have excessive lower anterior facial height.

Hyperdivergent Growth Pattern

Various longitudinal studies have been undertaken to identify and evaluate the facial and cranial patterns associated with the vertical development of face and the morphological features that are associated with the hyperdivergent facial pattern. Several linear and angular dimensions have been studied pertaining to the vertical

development of the face. The vertical development of the facial skeleton has been related to many skeletal units; the nasomaxillary complex, the alveolar processes, and the mandible and all have been associated with normal and abnormal vertical development.

Hellman (1931) suggested that a short ramus and a corpus rather than the vertical development in nasomaxillary complex leads to the development of open bite. However Hapak (1964), Subtelny (1964), Schendel (1976) also noted a steep mandibular plane and a large gonial angle in patients with hyperdivergent facial pattern. Several studies have demonstrated that total anterior face height is relatively large in persons with open bite faces and on the other hand, several investigators have also suggested that one of the key factors contributing to the open bite morphology is a reduction in the posterior height of the face. Sassouni and Schudy concluded that instead of total anterior facial height, the lower anterior facial height plays a dominant role in determining the vertical growth of the face. Hence, individuals with long face have much larger lower anterior facial dimension when compared to individuals with short faces.

Schendel and his associates described the 'long face syndrome' in those individuals who have excessive vertical growth of maxilla. Several investigations have also confirmed that the increased angle of the mandibular plane commonly found in persons with long faces is associated with a backward rotational growth pattern that can affect the vertical proportions of the anterior component of the face.

Thus, the hyperdivergent growth pattern may have several morphological features which may be of diagnostic value and these discordant dimensions produce a cumulative effect that results in an excessive anterior face height. According to many investigators, this aberrant facial growth pattern is established very early in life and it persists through out. Moss (1971), in a logarithmic spiral study of the locations of the foramen ovale and mandibular and mental foramina found that they were positioned more inferiorly very early in the facial growth of open bite cases. With continued development, inferior growth movements of the foramina retained these lower locations, so the distance between the foramen ovale and the mandibular foramen was shorter in open bite than with normal occlusions.

Enlow and Trouton (1983) conducted a radiographic study to evaluate the cranial and facial pattern that relate to the composite anatomical relationships that are associated with skeletal open bite and deep bite. They put forth the counterpart principle, to describe the growth pattern of craniofacial skeleton. The *counterpart principle* states that some of the principle skeletal parts of the skull are related to other specific parts in appropriate dimensions and placement. For example, the bony mandibular arch is a structural counterpart of the maxillary arch. If there is a dimensional or positional difference between them, a corresponding measurable misfit will be found. Various regional part/counterpart relationships involve major boundaries which are coincident with key sites to growth and remodeling. Two fundamental factors are involved in the evaluation of the part/counterpart relationship pertaining to vertical development of face. First, linear dimensions are compared and then the inclination (angulation, tilt, cant or rotational position) is determined. Following are the characteristic morphological features associated with hyperdivergent growth pattern.

Ramus Inclination

The ramus is the horizontal counterpart of the middle cranial fossa, a more backward position of the ramus may be expected to produce anterior open bite (Fig. 14.1).

Middle Cranial Fossa Inclination

The anteroinferior inclination of this part of the basicranium affects the placement of the nasomaxillary complex relative to the mandible. A greater forward-downward inclination of middle cranial fossa may be expected in deep bite while posterosuperior or more upright inclination of middle cranial fossa results in the corresponding positional deviations that are associated with open bite.

Posterior Maxillary Height

If the posterior part of the nasomaxillary complex is long vertically with respect to its counterparts, which is the combined heights of the ramus and middle cranial fossa, a relative downward and backward relationship of the entire mandible and open bite will be expected.

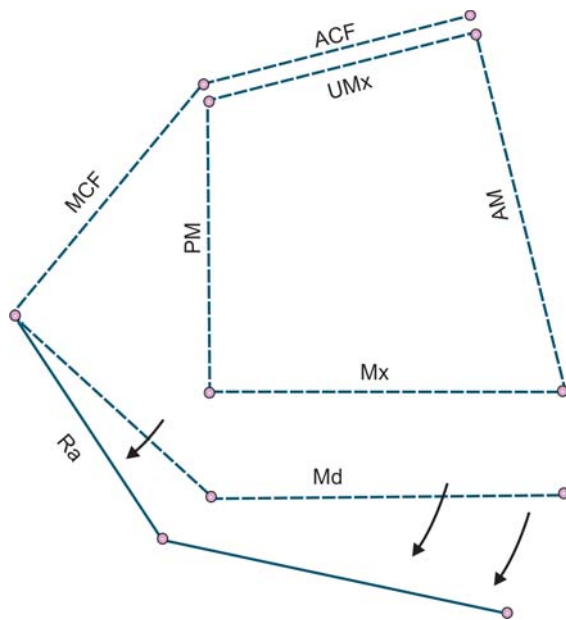


Fig. 14.1: A postero-inferior inclination of the mandibular ramus and corpus contributes to an anterior open bite. ACF: anterior cranial fossa; MCF: middle cranial fossa; UMx: upper maxillary region; PM: posterior part of maxilla (vertical); AM: anterior part of maxilla; Mx: maxillary arch; Ra: ramus; Md: mandibular arch (Source: Trouten JC. Morphologic factors in open-bite and deep bite. Angle Orthod 1983;53:192-211)

Horizontal Maxillary Inclination

An anterosuperior tilt of the maxillary alveolar process or the anterosuperior tilt of the palatal plane might contribute to the skeletal open bite tendency (Fig. 14.2).

Mandibular Plane Inclination

A downwardly inclined mandibular plane can contribute to the development of open bite. Besides this, a horizontally long mandible, a more open gonial angle, a lack of compensating curve of spee can contribute to open bite malocclusion (Fig. 14.3).

Enlow further stated that the growth activity in one region is invariably accompanied by complementary growth in other regions. This complementary activity is essential for maintaining functional and esthetic balance. Thus, if the anterior facial height is long, facial balance is preserved provided there is complementary growth activity in posterior facial height and mandibular ramus height. On the other hand, the growth pattern is

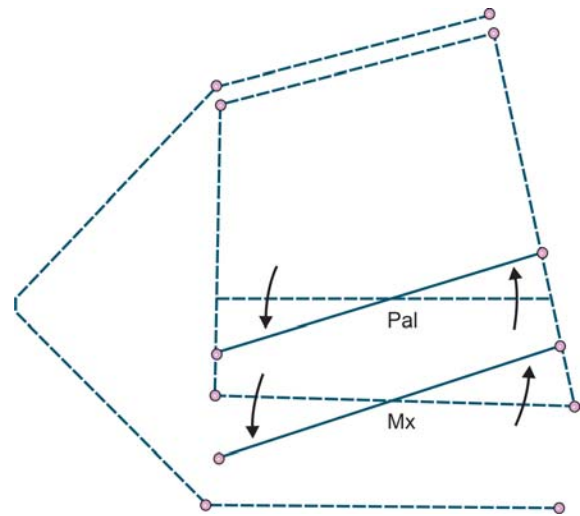


Fig. 14.2: Anterosuperior inclination of the palate and maxillary alveolar arch contribute to an anterior open bite (Source: Trouten JC. Morphologic factors in open bite and deep bite. Angle Orthod 1983;53:192-211)

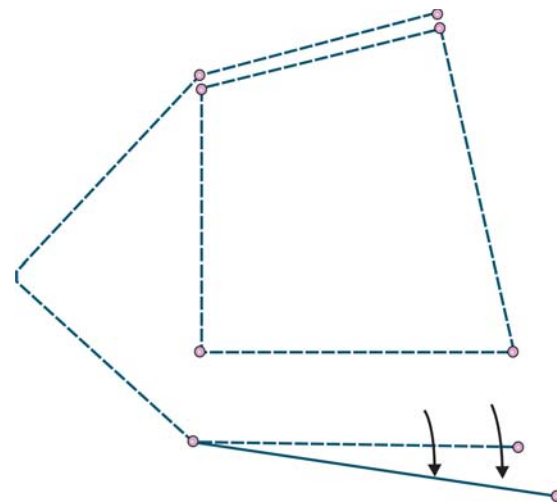


Fig. 14.3: A horizontally long mandibular arch, opening of the gonial angle and lack of a compensating curve of spee contribute to an anterior open bite (Source: Trouten JC. Morphologic factors in open bite and deep bite. Angle Orthod 1983;53:192-211)

disturbed in individuals with short posterior facial height leading to a tendency towards a skeletal open bite and disturbance in facial proportionality.

Similarly if the maxilla were rotated down posteriorly, a long ramus and acute gonial angle would compensate and allow normal facial proportions, but even a slightly

short ramus would produce downward-backward mandibular rotation and a long face open bite tendency. Thus, Enlow stresses the importance of the complementary growth of facial skeleton to preserve the facial harmony.

According to Schudy (1964), the hyperdivergent growth pattern of the jaws often results due to disharmony between vertical growth and anteroposterior or horizontal growth of jaws. He describes this as “clockwise rotation” of mandible wherein the vertical growth in the molar region is greater than that at the condyles, and the mandible rotates clockwise resulting in more anterior facial height and less horizontal change of the chin. Extremes of this condition cause open bites. Thus, it can be said that condylar growth is pitted against the combined vertical elements of growth. The final vector of growth of the chin is a resultant of the struggle between horizontal growth and vertical growth, in other words, between condylar growth and vertical elements. These vertical components are:

- Growth at nasion and in the corpus of the maxilla which produces an increase in the distance from nasion to anterior nasal spine and causes the maxillary molars and posterior nasal spine to move away from the sella-nasion plane;
- Growth of the maxillary posterior alveolar processes causing the molar teeth to move away from the palatal plane; and
- Growth at the mandibular posterior alveolar processes causing the molar teeth to move occlusally.

Besides this, the dorsal migration of the glenoid fossa is also a potent factor as it tends to cancel out the growth of the condyles; thus, in a sense, it is arrayed on the side of vertical growth.

Thus, clockwise rotation of the mandible is a result of more posterior vertical growth than condylar growth, the point of rotation being the condyles. As the vertical growth exceeds horizontal growth (condylar growth), pogonion cannot keep pace with the forward growth of the upper face and the mandibular plane becomes steeper, rotates backward often with a skeletal class II tendency. Bjork using implant studies described two types of backward rotation. For a detailed description about the rotations and its effect, refer Chapter 13 on rotations.

Isaacson (1971) stated that extremely high SN-MP angles are the result of relatively small amounts of vertical condylar growth and relatively large amount of vertical alveolar and sutural growth producing backward rotation of mandible. He also stressed the importance of the glenoid fossa position in determining the divergence of the face. Superior positioning of fossa produces the same effect as a short ramus.

It is commonly believed that hyperdivergent growth pattern is the reflection of unfavorable growth pattern. The features of hyperdivergent face are given in Table 14.1.

Hypodivergent Facial Growth Pattern

The hypodivergent face has been called by so many names: “forward rotation” (Bjork), “counterclockwise

Table 14.1: Features of hyperdivergent face

<i>Extraoral features</i>	<i>Intraoral features</i>	<i>Cephalometric findings</i>
• Dolicocephalic head • Leptoprosopic face • Long sloping forehead with heavy glabella • Long and thin nose • Large gonial angle • Short ramus • Long anterior face height • Short posterior face height • Downward and backward position of mandible • Convex soft tissue profile • Vertical mandibular growth • Ectomorphy • Weak temporal muscles • Incompetent lips	• Open bite relationship • High and narrow arched palate • Arch length discrepancy • Over erupted incisors • Impacted third molars	• Prognathic maxilla • Retrognathic mandible • FMA > 28° • SN-MP > 32° • Small interincisal angle • Vertical mandibular growth

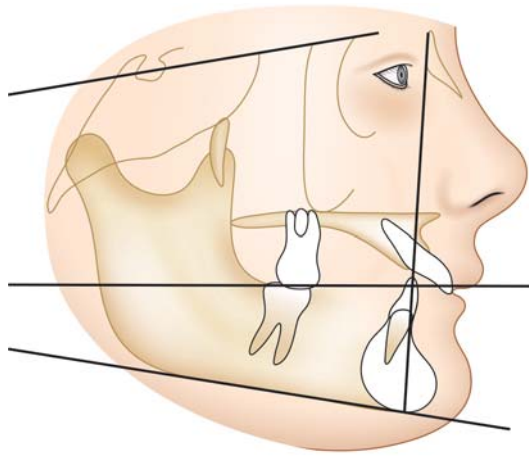


Fig. 14.4: A hypodivergent facial pattern where vertical growth is deficient

rotational pattern" (Schudy) or "Short face syndrome". It reveals the opposite end of spectrum of vertical facial growth. The resulting face is often squarish with a basal deep bite, and in such individuals the deep bite can be expected to get much worse with continuance of the growth pattern that is already present (Fig. 14.4).

Isaacson (1971) reported that mandibular displacement is transitory when the increments of vertical condylar growth equal the increments of vertical growth at the maxillary sutures and the maxillary and mandibular alveolar processes. However, if condylar growth exceeds the vertical growth at the sutural-alveolar process area, a forward or closing mandibular rotation would occur.

Schudy (1964) describes such counterclockwise rotation of the mandible as a result of more condylar growth activity than combined vertical growth in the regions of nasion, posterior alveolar process of maxilla and mandible. This type of rotation is nearly always accompanied by a forward movement of pogonion and "flattening" of the mandibular plane, which tends to increase the vertical overbite. Thus, the condyles grow out of proportion in the vertical plane leading to the closure of the SN-MP angulations.

The size of the gonial angle has an important influence upon the degree of resultant counter clockwise rotation. The smaller the gonion angle, the greater the rotation produced for each millimeter of forward movement of pogonion. When this angle is extremely small, it results

in extreme flattening of the mandibular angle together with the forward growth of the pogonion.

Bjork (1969) describes this condition as forward growth rotation and describes three basic types based on the centers of rotation (Refer chapter 13 on rotations).

Enlow and Trouton (1983) tried to identify certain regional counterparts, which in particular combinations might account for the composite, multifactorial morphological relationships that account for the hypodivergent growth pattern of the mandible.

- *Ramus inclination:* A more forward inclination of the ramus is expected to produce basal deep bite.
- *Middle cranial fossa inclination:* The anteroinferior inclination of this part of the basicranium affects the placement of the nasomaxillary complex relative to the mandible. A greater forward-downward inclination of the middle cranial fossa may be expected in deep bite (Fig. 14.5).
- *Posterior maxillary height:* If the maxilla is vertically short relative to the ramus and middle cranial fossa, a forward and upward rotation of the mandible ensues resulting in basal deep bite (Fig. 14.6).
- *Horizontal inclination of the maxillary plane:* An anteroinferior inclination of the maxillary alveolar process or the palatal plane results in deep bite (Fig. 14.7).

Besides these, an upwardly inclined mandibular plane, a horizontally short mandible, a more closed gonial

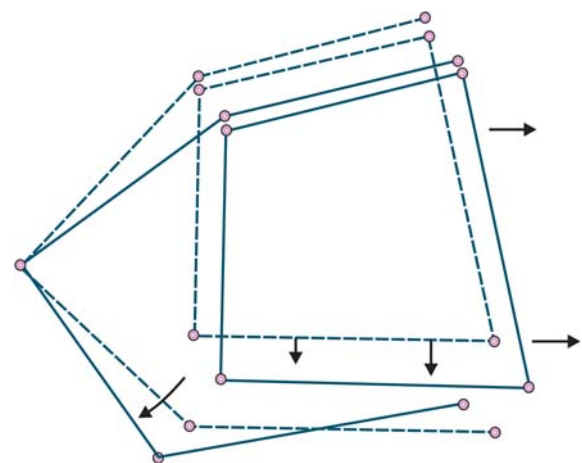


Fig. 14.5: A marked anteroinferior inclination of the middle cranial fossa and closure of the gonial angle contribute to deep bite (Source: Trouton JC. Morphologic factors in open bite and deep bite. Angle Orthod 1983;53:192-211)

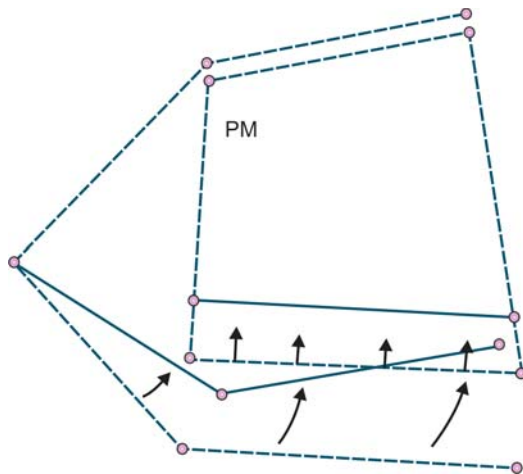


Fig. 14.6: A vertically short nasomaxillary (PM) dimension contributes to deep bite

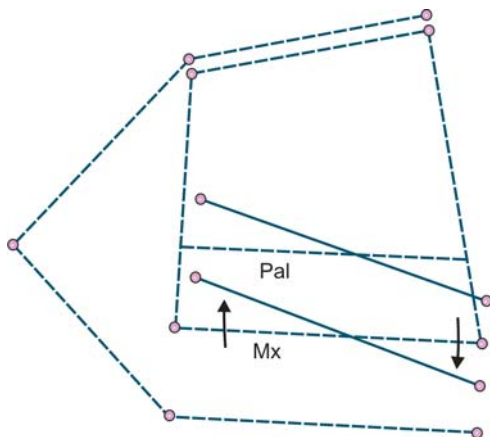


Fig. 14.7: Anteroinferior inclination of the palate (Pal) and maxillary alveolar arch (Mx) contribute to deep bite (*Source:* Trouten JC. Morphologic factors in open bite and deep bite. Angle Orthod 1983;53:192-211)

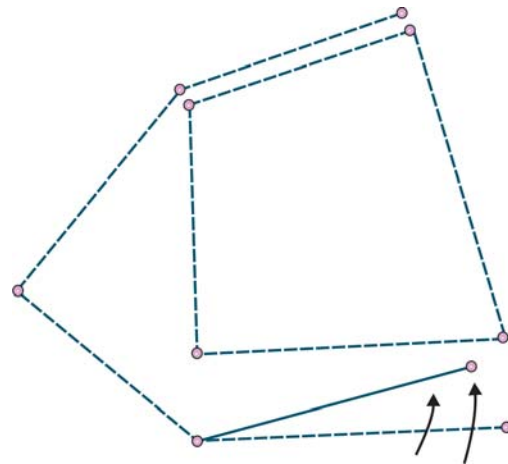


Fig. 14.8: A mandibular arch that is horizontally short relative to the maxillary arch, closure of the gonial angle and superior drifting of the mandibular anterior teeth (deep curve of spee) contribute to a deep bite

angle could contribute to deep bite malocclusion (Fig. 14.8). Features of hypodivergent face are given in Table 14.2.

GROWTH PATTERN OF SKELETAL CLASS II MALOCCLUSION

Class II Division 1 Malocclusion

The Class II malocclusion is characterized as skeletal when there is involvement of the jaws, and dental when there is just a dentoalveolar involvement; however, a combination of skeletal and dental factors have also been reported frequently. This malocclusion can be related

Table 14.2: Features of hypodivergent face

Extraoral features	Intraoral features	Cephalometric findings
• Brachycephalic head form • Euryprosopic facial form • Wide set eyes • Prominent cheek bones • Low mandibular plane angle • Horizontal or orthognathic growth pattern • Prominent chin • Straight or dished in soft tissue profile • Small gonial angle • Large and broad ramus • Reduced lower facial height • Thin lips • Strong masseter muscles	• Flat palatal plane • Broad arch • Crowded mandibular incisors • Deep overbite	• FMA < 25° • SN to MP < 32°

to a retrognathic mandible, prognathic maxilla, or a combination of both. Retro-positioning of the mandible might also be responsible for class II skeletal pattern. Understanding the morphology is a key element in planning dentofacial orthodontic treatment. Besides, this class II malocclusion is frequently complicated in the vertical plane of space, associated with skeletal open bite or deep bite, which play a significant role in determining the prognosis of orthodontic therapy.

The widely accepted clinical term “skeletal class II” does not specify whether the mandible is retruded in relation to the maxilla, or whether the maxilla is protruded in relation to the mandible. The class II malocclusions have a strong hereditary component as the etiologic factor, both in families and in ethnic and racial groups. The ethnic aspect also plays an important characteristic in the morphologic variation of these malocclusions. The complex etiology and great variety of morphologic and functional aspects of this malocclusion had motivated lots of cephalometric studies, both longitudinal and cross-sectional, to ascertain the growth pattern of craniofacial structure in class II malocclusions. The findings from the literature review

are still inconclusive regarding the dentofacial characteristics of class II division 1. The opinions of leading orthodontic researchers are controversial. Certain postulates had been made concerning the morphological differences between excellent occlusion and skeletal class II malocclusions, some of which include:

- Hellman (1922) working on skulls, concluded that class II, division 1 malocclusion skulls exhibited a more acute gonial angle than skulls with excellent occlusion. He postulated that this would account for the mandible being in a more distal relationship to the maxilla. In 1931, he repeated this investigation on living subjects, using anthropometric instruments. In this study, he concluded that the mandible is often subnormal in size, but always more posteriorly positioned when compared to cases of excellent occlusions.
- Renfroe in 1941 compared the facial pattern of individuals with class I and class II division 1 malocclusions using means of angular measurements (Fig. 14.9). He concluded that the mandible is not under developed, but is more posteriorly positioned and that the gonial angle is smaller in

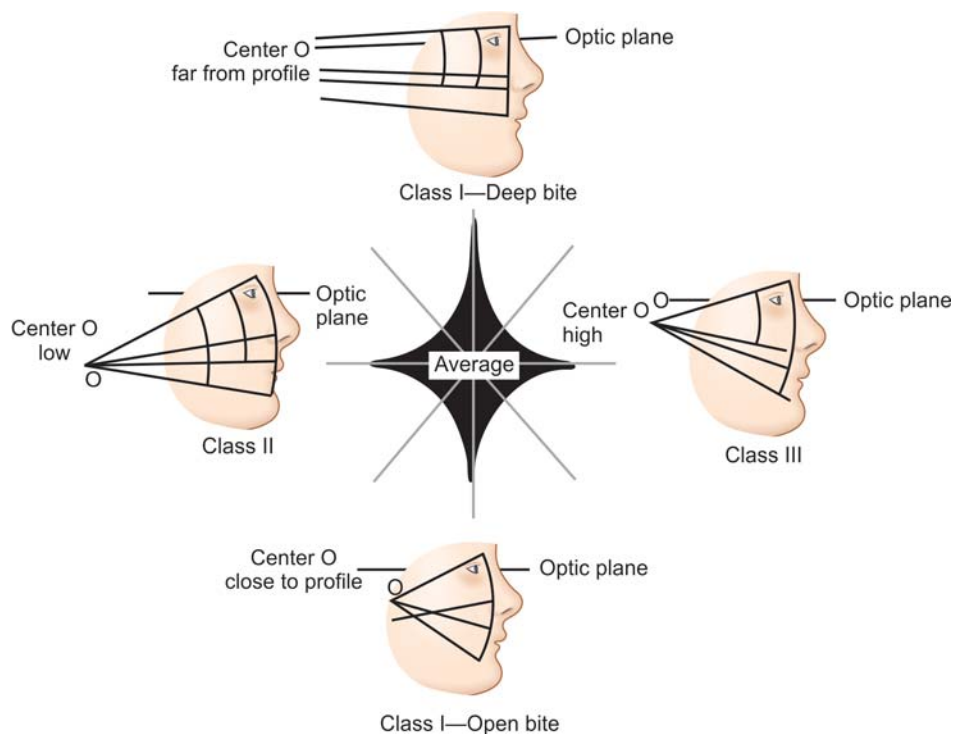


Fig. 14.9: Classification of facial types

class II division 1 malocclusions when compared to class I malocclusions.

- Elsassner and Wylie (1948) interpreted their findings to show that class II division 1 cases are a result of maxillary overdevelopment in males and mandibular underdevelopment in females, when the means were compared. They found the maxillary length to be greater in class II division 1 males, while in class II division 1 females the overall mandibular length was found to be less than in class I cases.
- Gilmore (1950) conducted a comparative cephalometric study on patients with excellent occlusions and class II division 1 malocclusions. Various linear and angular cephalometric measurements were made and compared. He concluded that the mandible in class II division 1 malocclusion is significantly smaller than the mandible in excellent occlusion group and he also found no significant difference in the size of the anterior cranial base either in males and females in class II division 1 malocclusion and in patients with excellent occlusion.
- Fisk (1953) described the following six morphological variations, characteristic of class II malocclusions:
 - The maxilla is anteriorly situated in relationship to the cranium
 - The maxillary teeth are anteriorly positioned in relationship to the cranium.
 - The mandible is of normal size, but posteriorly positioned.
 - The mandible is underdeveloped.
 - The mandibular teeth are posteriorly placed on a mandible which is normally positioned.
 - Various combinations of the above relationships.
- Sassouni (1970) describes several morphological patterns associated with class II malocclusion, which he calls the “Class II Syndrome”. This includes the combination of vertical and anteroposterior variations, which establishes four types of class II malocclusion.
 - Two types of deep bite, one with mandibular retrusion and one with maxillary protrusion.
 - Two types of open bite, one with mandibular retrusion and one with maxillary protrusion.
- Enlow (1971) tried to explain the craniofacial form and growth pattern of various individuals based on counterpart principle. By comparing the dimensions, angular relationships and growth changes for each of the many basic anatomic parts with their

respective counterparts, the craniofacial form and growth patterns of any given individual can be meaningfully appraised and the actual anatomic basis for them could be explained. If these regional counterparts are well balanced both in linear and angular measurements, the resulting face would be well balanced. However, anatomic misfit between various regional counterparts can result in skeletal malocclusions.

According to Enlow maxillary protrusion effect is produced under following conditions:

- A long maxillary arch.
- Short horizontal ramus or long posterior cranial fossa (PCF) dimension.
- Long vertical posterior nasomaxillary dimension or short composite ramus/PCF vertical dimension.
- Posterior direction of ramus alignment.
- Forward and downward alignment of PCF.
- Upward alignment of mandibular corpus and occlusion.
- Moyers (1980), by means of computer based statistical methods, collected a sample of 208 digitized cephalometric radiographs of children with class II malocclusion. He discovered several types of class II malocclusion with defining horizontal and vertical characteristics. He separated skeletal and dental factors responsible for class II malocclusions. Moyers describes six horizontal patterns and five vertical patterns of growth in class II division 1 malocclusions. According to him, several combinations of the horizontal and vertical types can occur in patients leading to different patterns of facial growth in class II patients.

Horizontal Types

Type A: It is characterized by a normal skeletal profile and normal AP position of jaws. There is maxillary dental protraction.

Type B: Displays midface prominence with normal mandible.

Type C: Displays class II profile; maxilla and mandible are retrognathic with protruded maxillary dental arch.

Type D: Displays retrognathic mandible, maxillary dental protraction.

Type E: Maxillary prognathism and bimaxillary dental protraction are features of this type.

Type F: Combination.

Vertical Types

Type 1: In this anterior facial height is greater than the posterior facial height.

Type 2: Is a square type of face. The skeletal deep bite with all the planes like mandibular, occlusal and palatal is more horizontal than normal.

Type 3: The palatal plane is tipped upward with decreased anterior upper facial height and resultant open bite.

Type 4: All the planes like mandibular, occlusal and palatal are tipped downward with the lip line unusually high on the maxillary alveolar process. Gonial angle is obtuse.

Type 5: Only the palatal plane is tipped downward, the occlusal and mandibular planes are normal. Gonial angle is smaller. Skeletal deep bite results.

- McNamara (1981) revealed that though class II malocclusions can result from several combinations of skeletal and dental factors, the retrusion of the mandible is the most common cause for skeletal class II malocclusion. Maxilla was found to be retrusive rather than protrusive in most cases of skeletal class II malocclusions and excessive vertical skeletal development was also a most frequent finding associated with retrusive mandible. Several other scientific investigations also proved that a lack of mandibular growth as the most prevalent type of retrognathism.

Class II Division 2 Malocclusion

The Angle's class II division 2 malocclusion is relatively rare when compared to division 1 malocclusion. The class II division 2 groups represent a significantly distinct population. Epidemiological investigations have shown that between 2 to 5 percent of the individuals in a population have Angle's class II division 2 malocclusions. Angle's definition is based on the clinical presentation of the dentoalveolar pattern. Physiognomically it may seem that patients with Angle's division 2 have some common traits like retroclined central incisors, deep bite, high lip line with deepened mentolabial sulcus and a prominent chin. However, it can be associated with greatly dissimilar types of craniofacial morphology.

A number of cephalometric studies were conducted to identify whether patients demonstrating clinical class II division 2 malocclusion have an underlying pathognomonic skeletal as well as dentoalveolar pattern.

Blair (1954) suggested that division 2 patients have more acute gonial angle, a decreased effective length of mandible and a more forward position of anterior outlines of maxilla and mandible. According to Renfroe (1948) the mandibular retrognathia in class II division 2 was total; i.e. it involved not only the B point and the chin but also gonion and the condyles. Besides the retrognathic pattern of mandible, a more consistent finding is that division 2 patients are often associated with deep bite. This is often skeletal, these individuals often show an upward and forward condylar rotational pattern with a short anterior face height. Wallis (1963) suggested that class II division 2 patients are distinct groups and these people showed longer anterior cranial base lengths, more acute gonial and mandibular planes and decreased anterior facial height and deep bite.

Cephalometric study conducted by Arnon et al (2001) has also concluded the following characteristic finding in class II division 2 malocclusions:

- The maxillary length is often normal.
- The mandibular length is shorter, and its sagittal position is retruded.
- The chin is prominent.
- The posterior facial height is definitely enlarged.
- The mandibular growth vector is horizontally oriented, and the mandibular plane is flat, creating the appearance of a hypodivergent facial pattern.
- The gonial angle is acute.
- The overbite is deep, probably due to extreme skeletal mandibular counterclockwise rotation rather than dentoalveolar over-eruption.

Cranial Base Flexure and Class II Malocclusions

Enlow et al (1971) and Enlow and McNamara (1973) stated that the cranial floor is the foundation on which the human face develops and demonstrated that the dimensions of the middle cranial fossa considerably influences the relationship between the nasomaxillary complex and the mandible. According to them, the positioning and the relative proportions of the facial parts with respect to the anterior and posterior segments of the cranial floor account for several basic characteristic types of facial features. A more open cranial base flexure during growth often occur in dolichocephalic faces, in which the midface is positioned more anteriorly, the

mandible is rotated downward and backward often resulting in class II malocclusion (Fig. 14.10).

Andreson and Popovich (1989) have also noted that in class II children, the jaws, especially the mandible, had a more posterior position under the cranium, and there was a more open flexure of the cranial base and shorter lower cranial height.

Of all the several combinations of morphological features that might be associated with class II malocclusions, retrognathic mandible associated with decreased total mandibular length is the most predominant growth pattern. This anteroposterior discrepancy between the maxilla and mandible is established early and class II dental relationship is maintained even though growth has improved the skeletal mandibular retrusion. However, assessing the growth pattern of skeletal class II in vertical plane is also important for assessing the prognosis for treatment. Forward mandibular rotation during growth is more favorable in the correction of class II malocclusions, on the other hand, backward rotation of the mandible during growth whose centre of rotation at the condyle or last occluding molars in general is not favorable in the treatment of class II malocclusions.

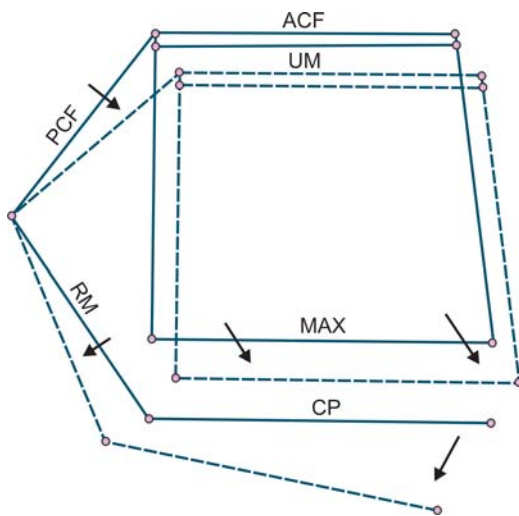


Fig. 14.10: Effects of cranial flexure on facial topography. Horizontal inclination of posterior part of anterior cranial base (PCF) relative to anterior cranial fossa (ACF) places the upper maxillary region (UM) and the maxillary arch (MAX) in a more protrusive position. It also lowers ramus (RM). This results in downward and backward rotation of the ramus and retrusion of mandibular corpus (*Source: Neurocranial basis for skeletal form and pattern—Enlow and McNamara. AO. 1973*)

GROWTH PATTERN OF CLASS III MALOCCLUSIONS

Skeletal class III malocclusions can be defined as a skeletal facial deformity characterized by a forward mandibular positioning with respect to the cranial base and/or maxilla. Numerous studies have been conducted to determine the morphologic variability of craniofacial complex in patients with class III malocclusion. These studies have shown that the term class III malocclusion is not a single diagnostic entity but can result from numerous combinations of skeletal and dentoalveolar components. This facial dysplasia can be classified into mandibular prognathism, maxillary retrognathism, or combinations of both, depending on the variation of the anteroposterior jaw relationships. The etiology and expression of a malocclusion must be understood before it can be clinically corrected.

Stapf (1948) subdivided class III deformities into typical type (exhibiting mandibular overgrowth) and atypical type (exhibiting a diminutive maxilla). He suggested that normal and class III malocclusions were hafted to the cranium orthognathically or retrognathically, and that craniofacial hafting determined the severity of class III deformities. He, thus, developed two theories; the first, that growth beyond normal limits leads to size changes and, second, that alterations in growth patterns lead to shape changes associated with a class III appearance concomitantly.

Guyer et al (1986) concluded that several morphological combinations, both anteroposterior and vertical were associated with class III malocclusions; simple maxillary retrusion was found in 25 percent of the cases while isolated mandibular protrusion was found only in 18.7 percent of the total sample; a combination of mandibular protrusion and maxillary retrusion was found in 22.2 percent of the sample. Guyer also concluded this aberrant growth pattern is often established early in life though it worsens in the later part of life.

Ellis and McNamara (1984) stated that the retrusive maxilla with prognathic mandible is the most common skeletal relationship accounting for 30 percent of all class III cases. Maxillary retrusion with normal mandibular prominence was found in 19.5 percent of the individuals and normal maxilla with mandibular protrusion was found in 19.1 percent of the individuals.

Enlow (1971) suggested the counterpart principle and growth equivalent theory to explain the aberrant

craniofacial pattern in various malocclusions. Abnormal sizes or positional malrelation is often attributed as disrupting factors in various malocclusions. According to him, a mandibular protrusion effect is produced in the following circumstances:

- A long mandibular arch.
- A long horizontal ramus relative to posterior cranial fossa (PCF).
- Short vertical nasomaxillary dimension or a long composite ramus/PCF vertical dimension.
- Anterior direction of ramus alignment.
- Downward alignment of the mandibular corpus and occlusion.
- Backward alignment or more upright PCF (Fig. 14.11).

Thus, skeletal class III does not represent a single entity of mandibular protrusion, rather a combination of aberrant growth pattern which includes several areas of the craniofacial skeleton including the cranial base, articulation with the glenoid fossa, size difference between the maxilla and mandible. Vertically, they can also be divided into two basic types depending on the vertical disproportions: long face and short face.

The aberrant growth behavior of the cranial base has also been strongly suggested in the possible etiology

of skeletal class III malocclusion. A size reduction of the anterior and posterior cranial base in class III anomalies was reported by several authors. Moss reported a smaller cranial base in association with class III malocclusion. The decreased posterior cranial base length and decreased angulations between the posterior and anterior cranial base, reflected by a closed cranial base angle is a more significant finding in skeletal class III malocclusion as it directly affects the positioning of the glenoid fossa. Enlow (1973) suggested that a more closed type of cranial base flexure during development, often seen in brachycephalic facial type, places the nasomaxillary complex in a more posterior and superior position and also aligns the mandible upward and forward often leading to class III malocclusion. According to Rakosi (1982), the prognathic pattern in class III began in cranial base area, the sella angle and articular angle were smaller in class III patients, moving the mandible anteriorly in relation to the cranial base. Sarnat (1983) also suggested that retardation of antero-posterior facial growth can be induced by a lag in the development of the cranial base.

Most cephalometric studies have also reported shortening of the posterior cranial base for class III patients as compared to class I and class II division 1 cases. Hopkins (1968) proved that the mean linear dimensions of cranial base show the smallest values in class III groups and the largest values in class II groups. Similarly, Dibbets et al (1996) noted a shortened posterior cranial base length in patients with class III malocclusion.

Morphometric and thin plate spline analysis, Singh et al (1997), to analyze the shape of the cranial base in subjects with class III malocclusion when compared with the normal class I configuration showed moderate deformations in the sphenoidal region of the mid-cranial base and significant changes affecting the occipital region of the cranial base, predominantly associated with the retention of a relatively acute cranial base angle, leading to deformation of the posterior cranial base as a significant component of class III malocclusion. They concluded that the deficient orthocephalization, or failure of the cranial base to flatten during development possibly plays an important role in the etiopathogenesis of class III malocclusions. Thus, a developmental disorder in the posterior cranial fossa area was suggested to account for the aberrant cranial base morphology in skeletal class III.

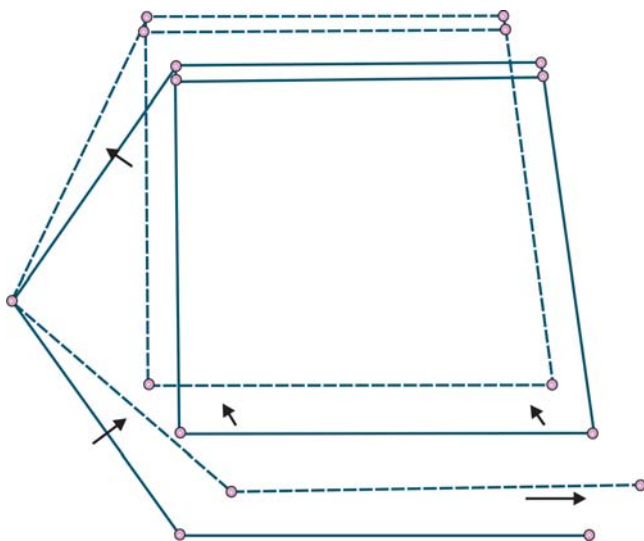


Fig. 14.11: Diagram represents a more upright alignment of PCF leading to superior and upward displacement of maxilla, forward and upward rotation of mandible (*Source: Neurocranial basis for skeletal form and pattern—Enlow and McNamara. AO. 1973*)

Besides the abnormal cranial base development of class III malocclusions, an increase of the sagittal mandibular length in association with a normally sized or shortened maxilla has been reported to be an invariable trait in class III anomalies in various cephalometric studies. An increase in mandibular length in class III individuals was substantiated by William and Anderson (1986), Mijiyama et al (1996) and many others.

Battagel (1993) undertook a retrospective study to identify the etiological factors underlying class III malocclusions and showed that the class III children showed differences in facial morphology in all facial areas examined, when compared with their control peers. The cranial base angle was more acute, the maxilla shorter and more retrusive, whilst the mandible was longer and more prominent. He also suggested that active growth of mandible continued even after puberty and class III females seemed to have a tendency towards horizontal development, whereas the males exhibited a more vertical growth pattern.

Singh et al (1998) used finite element analysis to analyze localized changes in size and shape of mandible in normal and class III individuals. They demonstrated that the differences between class III and class I mandibular configurations are due to a uniform increase in size locally, giving rise to a significant change in mandibular morphology. This increase in size (positive allometry) localized in the anterior extremity of the mandible may have a bearing upon mandibular prognathic appearance associated with class III malocclusions. These morphometric findings lend support to a developmental hypotheses of incremental condylar growth and mandibular allometry. With concomitant remodeling and absence of physical restraint, these developmental patterns may be associated with the development of mandibular prognathism. Thus, the anteroposterior growth of mandible associated with allometric enlargement of mandible during growth, which could be demonstrated even in the prepubertal period, might play an important role in the development of mandibular prognathism.

Thus, the aberrant growth pattern of mandible is also an important contributor in the development of class III malocclusions. Many studies have proved that the mandible continues to grow much larger and has a longer duration of growth when compared to normal occlusion.

One of the largest cross-sectional studies conducted by McNamara (2007) showed that significant mandibular changes occur until young adulthood (18 years on average), with increases between late maturation stages (4 through 6) that were twice as large as in subjects with normal occlusion for the class III females, and three times as large as in subjects with normal occlusion for the class III males. Growth trends toward accentuated class III profile and increased vertical dimension of the face also become apparent at late developmental stages (corresponding with complete eruption of the second and third molars).

Besides aberrant growth behavior of mandible, a significant percentage of class III individuals also show maxillary retrusion either alone or in combination with mandibular protrusion as an etiological factor in class III malocclusions.

Class III malocclusions are often complicated in the vertical plane. Some authors divide class III malocclusions into two basic morphologic types: divergent and convergent facial types. Excessive lower facial height was a rather frequent finding in adult patients with class III malocclusion. Chang et al (1992) and Guyer et al (1982) noted that the hyperdivergent class III pattern was not typically present in early childhood. Ellis and McNamara (1986) concluded that though the sagittal discrepancies between the maxilla and mandible is established early, a statistically significant increase in the lower anterior facial height was observed during the later stages of growth and is not typically present in early childhood. Similar studies conducted by McNamara et al (2006) also proved that an increase in lower facial height in class III individuals occurred in late developmental stages. Observing the vertical pattern of class III during growth is very essential in planning treatment. Hyperdivergent class III often represents a poor prognosis for orthopedic treatment. Class III malocclusions are often complex and the etiology is often genetic and familial occurrence has been documented in several studies.

SEXUAL DIMORPHISM IN VARIOUS MALOCCLUSIONS

One aspect of craniofacial growth that has received only limited attention is sexual dimorphism. According to Broadbent (1975) and co-workers, sexual dimorphism is the main feature in expression of secondary sexual characteristics that occur after puberty and during the

adolescent years. Most of the cephalometric variables that were analyzed in large-scale growth studies on Caucasian subjects presenting with a variety of malocclusions (The Bolton-Brush Growth Study, The University of Michigan Elementary and Secondary School Growth Study) exhibited significant differences between male and female subjects. Behrents (1993) did extensive longitudinal study in Caucasian population; various angular and linear measurements were compared. He concluded that the anterior cranial base was larger in males while the females showed more tendency towards horizontal growth. However, he could not document sexual dimorphism in sagittal positioning of maxilla and mandible, and dentoalveolar measures.

Jarabak (1984) conducted an epidemiological study to test for sexual dimorphism in various malocclusions and arrived at following conclusions:

- Majority of females demonstrate neutral pattern, whereas majority of males demonstrate hypo-divergent pattern.
- Sexual dimorphism is greatest in class II division 1 and class III.
- Males show a greater tendency towards prognathism, while females tend toward orthognathism and retrognathism.
- Mean values of all linear measurements are larger in males than females.
- Relatively strong correlations were found between various facial measurements like facial height ratio, gonial angle, mandibular plane angle, etc.

Significant degree of sexual dimorphism was noted in class III subjects than compared to other malocclusion groups. Baccetti et al (2004) showed that—(i) class III malocclusion is associated with a significant degree of sexual dimorphism in craniofacial growth especially after the age of 13 and (ii) female subjects with class III malocclusion present with significantly smaller linear dimensions in the maxilla, mandible, and anterior facial heights when compared with male subjects during the circumpubertal and post pubertal periods. Similar pattern of sexual dimorphism was also documented for other malocclusion groups. While few studies documented sexual dimorphism in various malocclusions, other studies failed to document such differences. On an average it was proved that the craniofacial complex is between 5 to 10 percent larger in males than females. Studies on dried skulls of males and females have also proved such

similar finding. This dimorphism was attributed to distinctly different pattern of maturational timing during pubertal growth. However, such studies on sexual dimorphism are limited and were conducted on only a few racial and ethnic groups.

POSITION OF GLENOID FOSSA IN DIFFERENT FACIAL TYPES

The positioning of glenoid fossa is directly dependent on the development of cranial base. The change in length of the posterior cranial base or cranial base flexure during development alters the spatial orientation of the fossa. Since the relationship of the mandible to the cranial base influences both sagittal and vertical facial disharmonies, the position of the glenoid fossa in relation to surrounding skeletal structures deserves a special mention. Altered positioning of glenoid fossa has been noted in different facial patterns and malocclusions.

Relative change in position of the glenoid fossa during facial development can occur as a result of local remodeling within the fossa or as a result of spatial repositioning of the entire temporal bone. Several investigators have evaluated the remodeling activity in the glenoid fossa following various types of orthodontic and/or orthopedic forces. Kokich (1987) noted that the posterior and inferior displacement of glenoid fossa takes place during growth which was mainly attributed to continued growth and remodeling at the circumtemporal articulations and secondarily to remodeling changes within the glenoid fossa itself. He also concludes that a more pronounced posterior displacement of fossa was predominantly seen in patients with vertical growth pattern.

A more posterior positioning of glenoid fossa was also associated in patients with class II malocclusions and retrognathic mandible. Various studies in the past have reported a tendency towards a skeletal class II pattern in subjects presenting with a large cranial base angle in association with a distal position of the temporo-mandibular joint within the skull. Rakosi stated that a non-compensated posterior positioning of the mandible caused by a large saddle angle is very difficult to influence with functional appliance therapy. Baccetti et al (2008), stated that a posterior positioning of glenoid fossa may be one of the diagnostic criteria in class II malocclusions.

On the contrary an anterior displacement of fossa during growth was noted in class III individuals. This was

attributed to a more upright posterior cranial base. Rakosi (1972) noted that saddle angle was decreased in class III individuals moving the mandible anteriorly. This coupled with excessive mandibular lengthening often worsened the class III condition.

Isaacson (1972) have also pointed out the role of the positioning of glenoid fossa in hypodivergent growth pattern. Glenoid fossa in these individuals is positioned more inferiorly which in turn has the effect of increasing the ramal length, which carries the mandible more forward and upward.

Thus, the relative positioning of the glenoid fossa during growth alters the growth pattern of the mandible. Baccetti (1997) studied the glenoid fossa positioning in different facial pattern and arrived at the following conclusion:

- Class II skeletal disharmony is associated with a more posterior position of the glenoid fossa when compared to class III skeletal disharmony.
- Subjects presenting with high angle vertical relationships show a more cranial position of the glenoid fossa and more caudal positioning of the fossa in horizontal growers.

Thus, growth of craniofacial skeleton is a complex and dynamic phenomenon involving various regions of the craniofacial skeleton. The constant interplay of genetics and environment often produce a mosaic of patterns in craniofacial growth. The external configuration of bone is dependent on a "composite of its functions" with remodeling and relocation often dependent upon its functional matrix. The various areas of craniofacial complex are, thus, subjected to different degrees of genetic and environmental influences during growth.

Disproportionate growth in any particular region of facial skeleton directly influences the orientation and spatial positioning of its counterpart. Extreme growth vectors in vertical and anteroposterior dimensions is often more challenging to orthodontists and drastically affects the treatment planning and prognosis of orthodontic mechanotherapy.

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Chapter 15

Growth and Craniofacial Anomalies

CHAPTER OUTLINE

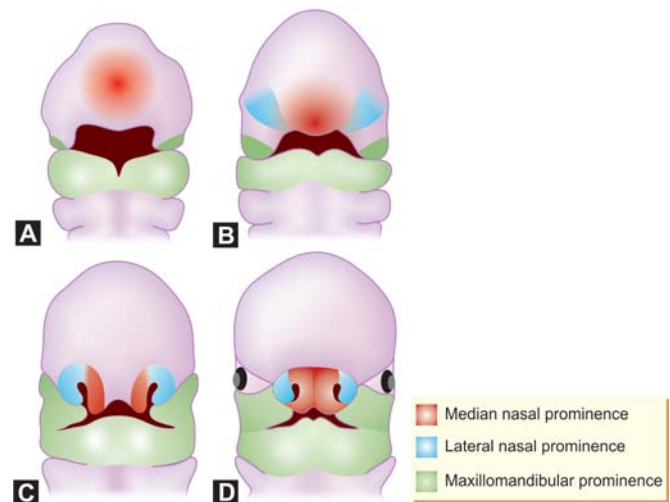
- Development of Craniofacial Primordia
- Neural Crest Cells
- Ganglionic Placodal Cells
- Patterning Branchial Arches in Head
- Homeobox Genes
- Developmental Anomalies
 - Retinoic acid syndrome
 - Thalidomide related craniofacial abnormalities
 - Neural tube defects
 - DiGeorge syndrome
 - Down's syndrome
 - Hemifacial microsomia
 - Treacher-Collin syndrome
 - Facial clefting
 - Achondroplasia
 - Premature closure of cranial and facial sutures
- Abnormal Growth Patterns

The development of head, face and neck is a complex phenomenon. The development of the facial structures starts as early as during the second week of intrauterine period. During this period of intrauterine development, the craniofacial skeleton is one of the earliest skeletal structures to be formed in the body. Disturbances to development during the early period of pregnancy frequently results in developmental anomalies of the head and neck. A thorough knowledge is required about the various regulating mechanisms governing craniofacial growth to understand the pathogenesis and management of craniofacial anomalies.

DEVELOPMENT OF CRANIOFACIAL PRIMORDIA

The craniofacial area is formed by five facial prominences namely the median frontonasal prominence, the two

maxillary and the mandibular prominences. The frontonasal process gives rise to the lateral nasal process and the median nasal process (Figs 15.1A to D). Both the maxillary and the mandibular processes are derived from the first branchial arches. The wide frontonasal process intervenes between the laterally developing eyes and contributes to the forehead and the nose. Specialized epithelial thickenings called olfactory or nasal placodes,



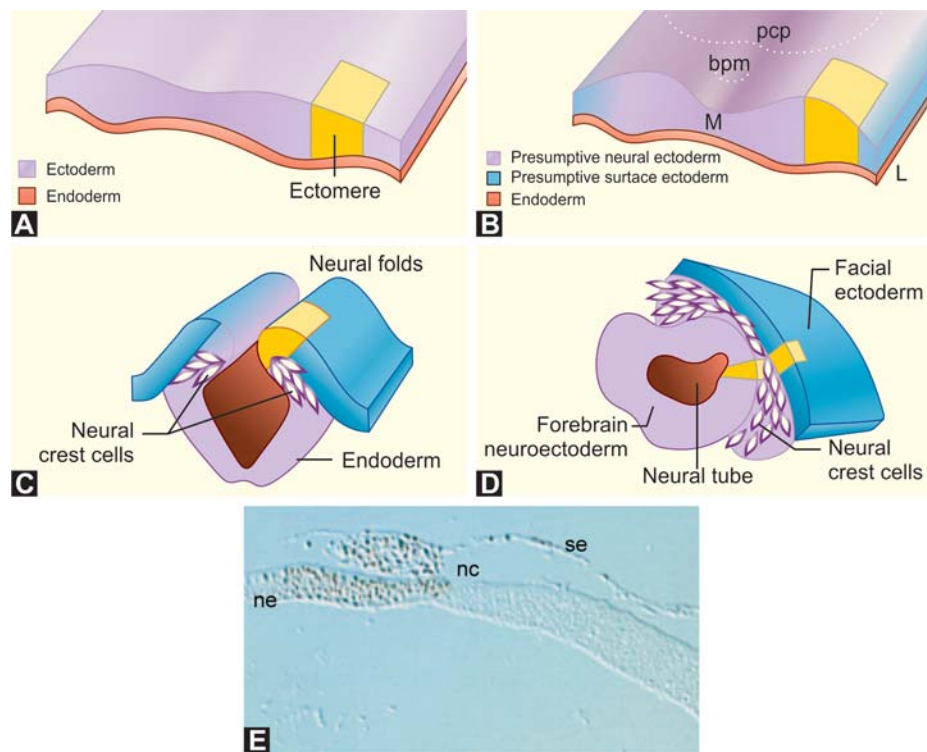
Figs 15.1A to D: Development of the craniofacial primordia. (A-D) A frontal view of the prominences that give rise to the main structures of face. The frontonasal (or median nasal) prominence (red) contribute to the forehead (A), the middle of the nose (B), the philtrum upper lip (C) and the primary palate (D), while the lateral nasal prominence (blue) forms the sides of the nose (B,D). The maxillomandibular prominences (green) give rise to the lower jaw (specifically from the mandibular prominences), to the sides of the middle and lower face, to the lateral borders of the lips, and to secondary palate (from the maxillary prominences)

form at inferolateral margins of frontonasal process which later sink to form the nasal pits. The lateral nasal process forms the ala of the nose. The medial nasal processes approach each other to form a single globular processes which in turn forms the tip of the nose, nasal septum, the columella, the philtrum and labial tuberculum of the upper lip, the frenulum and the entire primary palate. The maxillary process forms the major part of the upper jaw, the lateral part of the upper lip and the secondary palate. The mandibular processes of the two jaws unite to form the lower jaw.

NEURAL CREST CELLS

The neural crest cell is a highly pluripotent cell population that plays a critical role in development of vertebrate head. Development of the neural structures starts with

the infolding of the neural plate ectoderm along the midline forming the neural folds that fuse to form the neural tube which submerges beneath the superficial covering, the cutaneous ectoderm. This process is called neurulation (Figs 15.2A to E). In this process, the cells at the margins of the neural folds undergo an epithelial to mesenchymal transition following an inductive interaction between neural plate and presumptive ectoderm. This results in the formation of neural crest cells. Arising from the margins of the crests of the neural folds, the neural crest cells first appear in 7–14 somite stage embryos. This is fundamental in craniofacial growth. The differentiation, development, and migration of neural crest cells are crucial to craniofacial morphogenesis. The neural crest cells exhibit properties of both ectoderm and mesenchyme.



Figs 15.2A to E: Neurulation in the developing vertebrate embryo. (A) Neurulation begins with a unified layer of ectoderm, underneath which lies the endoderm. (B) The ectoderm begins to fold upwards, giving rise to the neural folds. During this process, interactions between signaling molecules begin to delineate the medial ectoderm as being neural (purple) and the lateral regions of ectoderm as being non-neural (blue). The prechordal plate mesendoderm (pcp) and the buccopharyngeal membrane (bpm) become evident at this stage. (C) The neural tube forms upon fusion of the neural folds, giving rise to discrete neuroectoderm (purple) and surface ectoderm (blue). Around the same time, the border region between the neuroectoderm and surface ectoderm gives rise to neural crest cells. The surface ectoderm and neuroectoderm of single ectomeres remain aligned during this process. (D) Neurulation completes upon formation of the neural tube, and neural crest cells (nc) lie sandwiched between the facial (surface) ectoderm and the neuroectoderm. Again, the individual neuroectoderm and surface ectoderm components of the ectomere remain in register. (E) Sagittal section through neural tube of a stage 15 chick embryo, showing neural crest (nc) located between surface ectoderm (se) and neuroectoderm (ne). L, lateral; M, medial

Characteristics of Neural Crest Cells

Unlike most parts of the body, the facial mesenchyme is derived principally from the neural crest and not the mesoderm of the embryonic germ layer. These neural crest cells migrate to the area of the branchial arches and give rise to the mesenchyme of the pharyngeal arches. The mesenchyme gives rise to the cartilage, bone and the muscles. The neural crest cells migrate extensively throughout the embryo in four overlapping domains. They are bipolar in configuration. Their elongated form is oriented in the direction of migration. Shape changes are noted and they also round up for cell division during migration. There is also a change in the cell surface receptors, from cell adhesion molecules to fibronectin, which permits make and break connections (Figs 15.3A and B).

The origin, migration and differentiation of the neural crest cells are mainly controlled by the homeobox Hox genes. Hox genes are responsible for the segmentation of the hind brain to eight segments called rhombomeres which is center to the subsequent development of neural crest mesenchyme. The Hox genes are expressed in a stepwise manner, delineating the cascading streams of ectomesenchyme that migrate from their dorsal origin to their ventral destination to create six pharyngeal arches and five facial prominences—the median frontonasal prominence, the paired maxillary and mandibular prominences bordering the central depression of the stomodeum. These facial swellings are the consequence of the neural crest ectomesenchyme invading the rostro-ventral aspect of the prosencephalon. The outgrowth

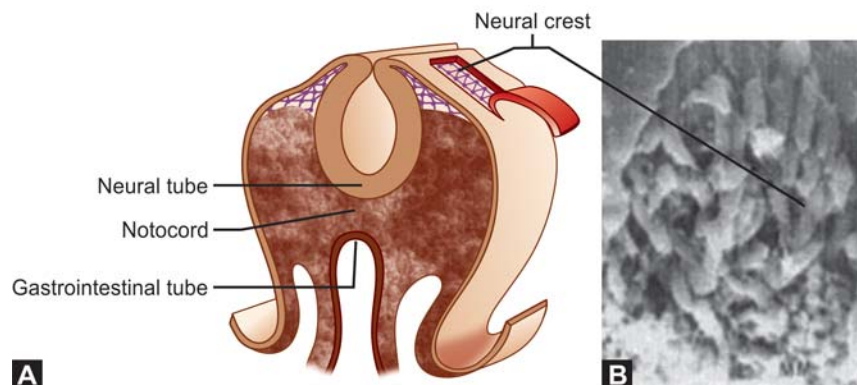
of the facial prominences is the result of the ectomesenchymal interactions with instructive signals emanating from each other.

Five growth factors control facial growth by regulation of cell proliferation, survival and apoptosis. These include endothelins, fibroblast growth factors (FGFs), sonic hedgehog (Shh), wingless (wnts) and bone morphogenetic proteins (BMPs). These factors are responsible for the ectomesenchymal interaction which is very important for origin, migration and differentiation of neural crest cells.

GANGLIONIC PLACODAL CELLS

Another interesting feature in craniofacial development is development of sensory neurons. In the trunk, all sensory neurons develop from the neural crest while in the head, the earliest differentiating neurons in the ganglia are derived from ganglionic placodes. These neurons constitute half of the total neurons. The significance of this dual origin is that they respond differently to neurotrophic factors such as NFG. Another difference is that the proprioceptive neurons are present in the central nervous system. There are five specific stages given by Johnston and Bronsky for embryonic craniofacial development:

- Germ layer formation and initial organization of craniofacial structures.
- Neural tube formation and interactions of cell population during initial formation of oropharynx.
- Origin, migration and interactions of cell populations, especially neural crest cells.



Figs 15.3A and B: (A) The onset of crest cell (diamond pattern) migration (arrows in A). The scanning EM (B) illustrates the morphology of mouse embryo crest cells during their migration (surface ectoderm peeled back as illustrated in Figure 15.3A). The morphology of crest cells is similar in all species studied. They are usually bipolar and oriented in the direction of migration (arrow in B)

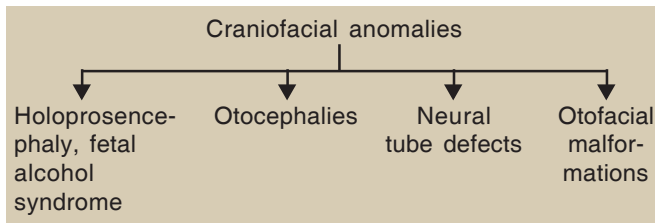
Box 15.1: Stages of development and related abnormalities

Stage	Time (humans) postfertilization	Related syndromes
Germ layer formation and initial organization of structures	Day 17	Fetal alcohol syndrome
Neural tube formation	Days 18-23	Anencephaly
Origin migration and interaction of cell populations, formation of organ systems	Day 19-28	Hemifacial microsomia Mandibulofacial dysostosis Limb abnormalities
Primary palate	Days 28-38	Cleft lip or palate, other facial clefts
Secondary palate	Days 42-55	Cleft palate
Final differentiation of tissues	Day 50-birth	Achondroplasia, synostosis syndromes

- Formation of organ systems, especially the pharyngeal arches and the primary and secondary palate.
- Final differentiation of tissues (skeletal, muscular and nervous elements).

Any disturbance in each stage will result in a specific type of abnormality (Box 15.1).

Another system of classification of craniofacial anomalies given by Jhonston and Bronsky is as follows:



PATTERNING BRANCHIAL ARCHES IN HEAD

Fundamental to the development of the craniofacial complex is the central nervous system. The central nervous system (CNS) arises from the neural plate, rolls up along its anterior, posterior axis to form the neural tube, and the enlarged anterior end divides into three vesicles. These vesicles are the primordial of the forebrain, (prosencephalon), midbrain (mesencephalon), and hindbrain (rhombencephalon). It is the rhombencephalic derived neural crest cells that give rise to the mesenchyme of the branchial arch. Migration of these populations of neural crest cells from the regions of the rhombencephalon results in a ventral location within the branchial arches. Development of the mid and the lower facial region is intimately associated with the branchial arches. It is clear that the neural crest cells derived from the hind brain is essential for normal formation of the face and neck. The hind brain is segmented into eight subunits

called rhombomeres, which have distinct morphological properties which vary with two segment periodicity. Each rhombomere is committed to a particular developmental fate. The neural crest cells that migrate and form the facial mesenchyme arise from the same level of the neural tube as the rhombomeres whose neurons will ultimately innervate the same facial mesenchyme.

Neural crest cells destined for the first branchial arch migrate from rhombomeres 1 and 2 while for the second and the third arches migrate from rhombomeres 4 and 6. The rhombomeres 2, 4, and 6 contain the exit point of V, VII and IX cranial nerves. Thus there is an axial specific code which exists before the migration of the neural crest cells.

HOMEBOX GENES

An excellent example of molecular studies as applied to the craniofacial embryogenesis is the homeobox gene. These were first discovered in fruit fly (*Drosophila*) and subsequently in other organisms. The genes in homeobox codes for transcription factors correlate with specific segments in the axial regions of the body of the embryo. The differentiation of neural crest mesenchymal precursors into facial tissues is regulated by Hox genes. The Hox genes that play a role in craniofacial development include, Hox A1, Hox A2, Hox B2, Hox B3, Hox B4, Msx1, Wnt1, etc.

Hox genes (Fig. 15.4) are expressed in the migrating neural crest cells from which the crest originates. Arch I is populated with crest from the posterior mesencephalon, and R1/R2. None of these express Hox genes. Arch II which is populated with crest from R4 and has minor contributions from R6 and R5 expresses Hox-2. Arch III is populated with R5 and R7; these cells

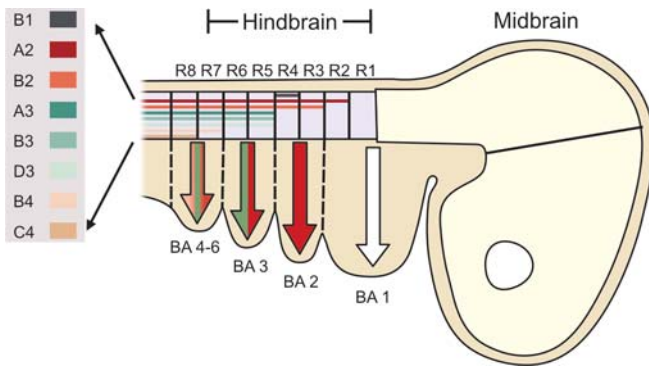
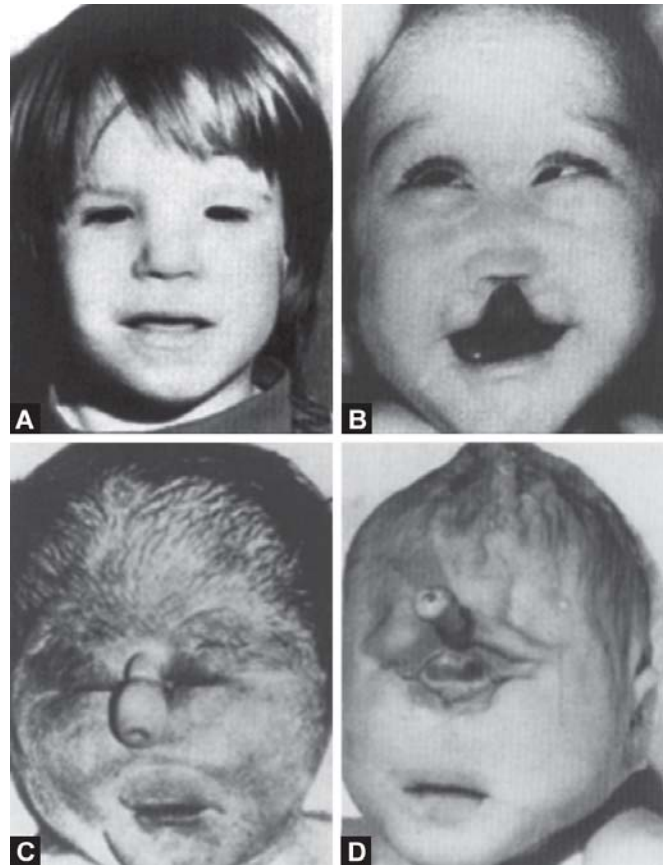


Fig. 15.4: Hox genes

express Hox-2 and Hox-3. Arch IV-VI form a poorly individualized group populated essentially by R7 and the crest expresses Hox-2 and Hox-3. Hox genes are not involved in the first branchial arch or more rostral head regions. There are other genes *Emx2*, *Otx-1*, and *Otx-2* patterning for the anterior regions of the head. A number of other genes are expressed in maxillary and mandibular arch and developing facial primordial arch. These include *MSX-1*, *MSX-2*, *DLX-6*, *BARX-1* and *ET-1*.

MSX-1 and *MSX-2* plays a special role in specification of skull and face. Disruption of *MSX-1* and *MSX-2* in experimental animals has led to loss of palatine shelves, absence of palatine bones, maxillary and mandibular hypoplasia and tooth agenesis. Defective expression of *MSX-2* causes defects in skull ossification due to defective osteoprogenitor proliferation during calvarial morphogenesis. *DLX-1* is another gene which regulates the formation of neural crest derived elements in maxillary and mandibular arches. It mainly controls the proximal arch structures.

Another family of homeobox genes is goosecoid. Endothelin-I (*ET-1*), a member of that family codes for a vasoactive peptide which play a role in regulation of blood pressure. Disruption of this gene in experimental animals produced cardiovascular abnormalities, reduction in tongue size, micrognathia and cleft palate. Sonic Hedgehog is another protein which is necessary for the patterning of the neural plate. Defective patterning of the neural plate results in holoprosencephaly, failure of cleavage in the midline forebrain and cyclopia. Sonic hedgehog, also expressed in the mesenchyme of the frontonasal process and maxillary process, is found to be necessary for their normal development (Figs 15.5A to D).



Figs 15.5A to D: (A to C) Represent holoprosencephalies of increasing severity. The child with the fetal alcohol syndrome (A) is a mild form, while arrhinencephaly (B) is of intermediate severity, and ethmocephaly (C) is the most severe expression. Eye size apparently decreases with increasing severity. The single large eye seen in cyclopia perfecta (D) probably arises by a somewhat different mechanism (see text). (A, from Sterling Clarren; B, from Ross and Johnston [1972]; C, from Taysi and Tinaztepe [1972]; and D, from Gorlin et al [1990])

DEVELOPMENTAL ANOMALIES

Abnormalities of Neural Crest Cell Origin and Migration

Holoprosencephaly and Fetal Alcohol Syndrome

It is characterized by decreased forebrain and increased tendency for the three ventricles to form a single cavity. The main defect is reduced midline components. Facial defects include defects of medial nasal prominence. Derivatives of the medial nasal process including philtrum and portions of maxilla (premaxilla) are deficient. Contact of olfactory placodes in the midline results in failure of

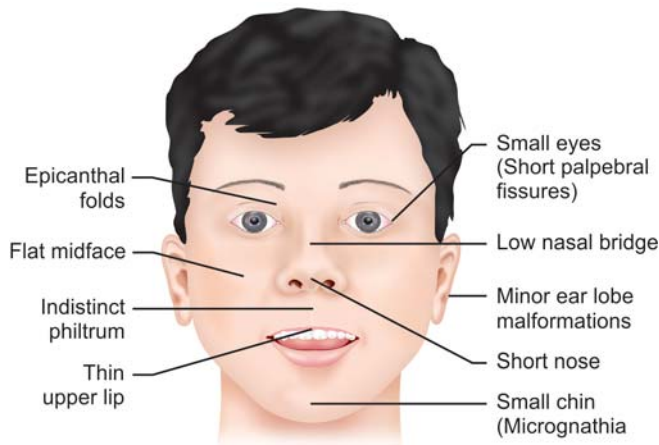


Fig. 15.6: Features of fetal alcohol syndrome

the medial nasal prominences to develop and leads to arrhinencephaly. Increasing deficiency leads to progressively smaller eyes which may unite to form one median eye or remain as two small eyes close to the midline. Exposure to high levels of ethanol at early stages of fetal development produces fetal alcohol syndrome (FAS) which now is recognized as one of the holoprosencephalies (Fig. 15.6).

Ethanol has direct effects on neural plate or the mesoderm. This results in considerable cell death in anterior neural plate. Normal programmed cell death is necessary for eliminating selected adult cell types. This takes place by apoptosis, which is required for normal sculpting of the embryo. If apoptosis becomes excessive, the embryo's ability to process the debris becomes overwhelmed and leads to abnormal development. The homeobox gene *MSX1* and *MSX2* are essential for the normal regulation of apoptosis.

Retinoic Acid Syndrome

This syndrome appeared after the introduction of the acne drug Acutane in 1982. Retinoic acid contains 13-cis-retinoic acid. The severity of the anomaly depends on the degree of metabolism of the drug. The levels of the metabolite 4-oxo-retinoic acid are 3 to 5 times higher than the original parent drug concentration and act as teratogen. The main target of retinoic acid is the neural crest cells. The neural crest cells are killed before leaving the neural plate which occurs at a later period. It was found that retinoic acid increases the expression of the *MSX2* and causes upregulation of retinoic acid

receptor β (*RAR β*), which in turn causes increased affinity for retinoic acid and further increased *MSX2* expression causes excessive apoptosis which causes loss in neural crest cells.

The clinical features of retinoic acid syndrome are:

- Microtia.
- Facial bone and calvarial abnormalities.
- Micrognathia.
- Cleft palate.
- Congenital heart disease.
- Aortic arch abnormalities.
- Cerebellar hypoplasia and vermis agenesis.
- Microcephaly.
- Limb abnormalities.

Thalidomide Related Craniofacial Abnormalities

Thalidomide was a drug sold in Germany extensively as an over-the-counter tranquilizer. Many of the early exposures produced craniofacial and cardiovascular malformations similar to retinoic acid. Depending on the time of exposure, it produced malformations similar to retinoic acid syndrome (exposure on 19-23 days) and related syndromes, as well as Treacher-Collin syndrome (exposure on days 25-26). Other manifestations include limb defects, both pre- and post-axial hypoplasia. Thalidomide poisoning also causes cleft of the palate.

Neural Tube Defects

It is one of the five most common human malformations. The problems are related to neural tube closure, principally neural fold elevation and contact. Neural tube defects are those involving the brain (anencephaly) and the spinal cord. Anencephalies are usually lethal. There are secondary facial abnormalities, of which occasional cleft palate (CP) is severe.

DiGeorge Syndrome

This syndrome is related to maternal alcoholism. The manifestations are similar to retinoic acid syndrome except for the short upper lip which is not seen in retinoic acid syndrome. A unique feature of this syndrome is the occurrence of pharyngeal gland problems (thyroid and parathyroid deficiencies). The main etiological agent is ethanol which is lethal for migrating neural crest cells. This syndrome is frequently associated with chromosomal deletion 22 (Fig. 15.7).

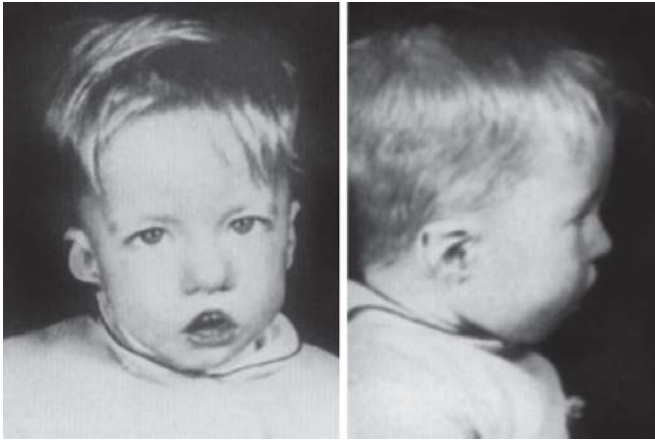


Fig. 15.7: Child with the DiGeorge syndrome. In addition to the external ear malformation, the mandible is somewhat underdeveloped. In contrast to the retinoic acid syndrome, the upper lip is short, particularly in its central portion. From Kretchmer et al (1968)

Down's Syndrome

It is a chromosomal disorder that occurs mainly due to trisomy 21. It can also occur due to translocation in which extrachromosomal material is translocated to chromosome G or D group and rarely due to chromosomal mosaicism.

Clinical features of Down's syndrome are flat face, larger anterior fontanelle, with open sutures, small slanting eyes with epicanthal folds, open mouth, frequent prognathism, sexual underdevelopment, cardiac abnormalities, and hypermobility of the joints. The clinical features, like the short upper lip in the midline, and a lop-ear are similar to those seen in DiGeorge syndrome. The main defect is in the migration of neuronal cells of cardiac mesenchyme.

Hemifacial Microsomia

It occurs in 1:4000 livebirths. It is a common otofacial malformation. It is frequently associated with conotruncal and vertebral abnormalities. There are no clear environmental associations. In most or all cases neural involvement is seen. It is characterized by a lack of tissue on the affected side of the face, usually in the area of the mandibular ramus and external ear (Fig. 15.8).

Poswillo in the 1970s suggested that hemorrhage from stapedial artery and tissue necrosis might be involved in the development of hemifacial microsomia.

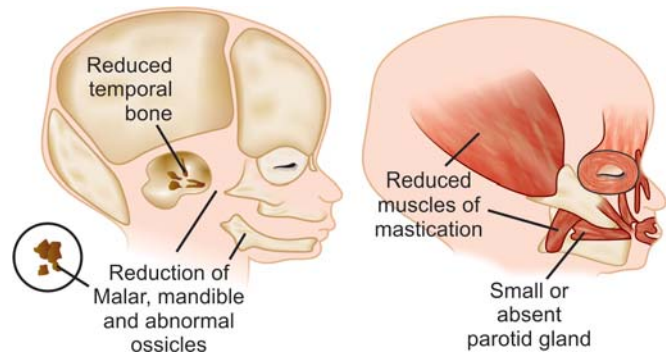


Fig. 15.8: Hemifacial microsomia. In addition to the malformation of the external ear, as seen in this patient, many regional structures are usually deficient (patient and diagram). These include the middle ear ossicles, squamous portion of the temporal bone, mandible, muscles of mastication, and the parotid gland. The malformations are often largely limited to one side, as in the patient illustrated [Poswillo (1973)]

Stapedial artery forms the temporary blood supply to the area of developing ear and mandibular ramus between 33rd and 40th day of gestation, which is later taken over by maxillary artery. The outer part of the stapedial artery atrophies and seals off. Poswillo suggested that hemorrhage from the stapedial artery causes facial defects associated with hemifacial microsomia. It was also found that hemifacial microsomia was associated with many defects resembling those arising from neural crest cell loss. Thalidomide produced many malformations with facial patterns similar to hemifacial microsomia. This suggested that variations in the naturally occurring hemifacial microsomia result from differing expressions of the same basic defect, early loss of neural crest cells. The main etiology is the death of neural crest cells with the longest migration path. Those taking circuitous route to the lateral and lower areas of the face are most

affected, whereas those going to the central face tend to complete their migratory movement. This explains why midline facial defects including clefts are rarely part of the syndrome.

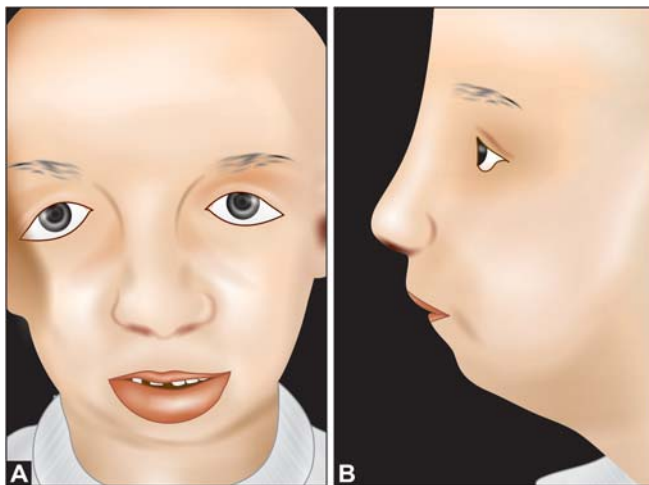
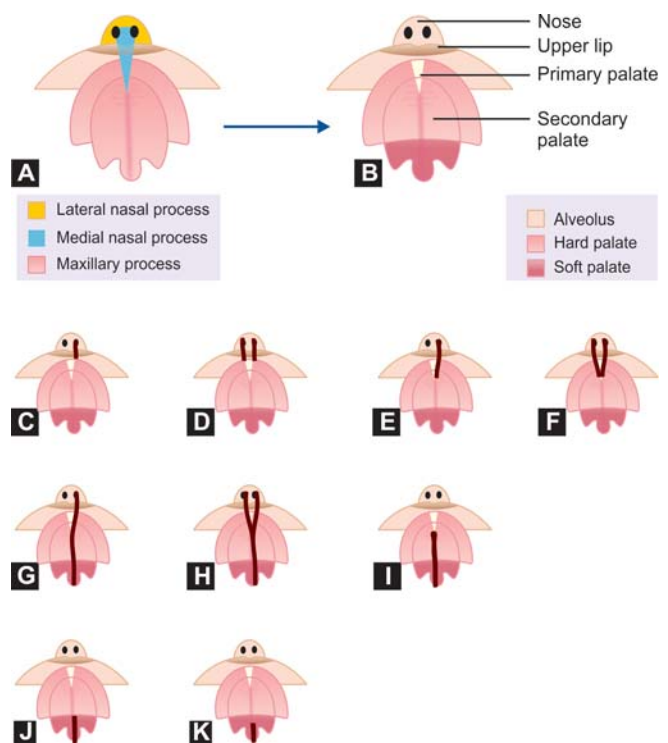
Treacher-Collins Syndrome (Figs 15.9A and B)

This was frequently called as first arch syndrome involving structures derived from first arch. It is characterized by bilateral deficiencies in the lateral orbital rim and zygomatic area in addition to absent or rudimentary mandibular condyles, short mandibular ramus, severe antegonial notching and retrognathia. The shape of the mandible with a markedly down-turned symphysis is a characteristic feature. Down-turned palpebral fissures, coloboma, missing eyelashes, aberrant facial hair over the malar area and ear deformities are likely to be present. The main problem is due to the massive cell death in the trigeminal ganglionic placode, which alters the further development of the placodal cells, ultimately resulting in secondary defects in neural crest cell derivatives.

Facial Clefting (Figs 15.10A to K)

The most prevalent congenital defect of dentofacial development is clefting of the lip and or palate, which occurs in approximately 0.1 percent of births in United States. The incidence of cleft lip in white population is 1:800 to 1000 livebirths, higher in Asian populations albeit 1:500 to 600 livebirths and lower in blacks about 1:2000 livebirths. The incidence of cleft palate is 1:2000 livebirths. Clefts are classified as syndromic (those

associated with other malformations such as holoprosencephalies, hemifacial microsomia and Treacher Collin's syndrome) and non-syndromic (clefts that are not associated with well defined syndromes). Clefts occur due to any disturbance in the fusion of the facial process namely the medial nasal process, lateral nasal process, maxillary process and the palatine shelves from the maxillary process. Cleft of lip occurs due to the failure of fusion between the median and lateral nasal process and the maxillary process which occurs during the 6th week of development. Midline cleft of the upper lip



Figs 15.9A and B: Treacher-Collin syndrome

Figs 15.10A to K: Embryological origins of the midline facial structures (A, B). In the developing embryo, the lateral nasal processes form the alae and sides of the nose, while the medial nasal processes form the intermaxillary segment, composed of the upper lip philtrum, the primary palate and the four incisor teeth. The maxillary process forms the remainder of the upper lip and the secondary palate, consisting of the hard palate and associated dentition anteriorly and posteriorly, and the soft palate. Various types of orofacial clefting: (C) unilateral cleft lip; (D) bilateral cleft lip; (E) unilateral cleft lip and primary palate; (F) bilateral cleft lip and primary palate; (G) complete unilateral cleft of the lip and palate; (H) complete bilateral cleft of the lip and palate; (I) isolated cleft of the secondary palate; (J) isolated cleft of the soft palate; (K) submucous cleft of the soft palate

occurs due to split within the median nasal process, but it is rare. Since fusion of lateral nasal process is accompanied by fusion of median nasal process, defects in fusion not only creates primary palate but also part of the alveolar ridge. Cleft lip is usually accompanied by a notch in alveolar ridge when the secondary palate is not involved.

Closure of the secondary palate follows that of the primary palate around 8th week of intrauterine life. Since closure of secondary palate follows the primary palate, interference with lip closure can also affect palate closure. An isolated cleft palate occurs after the lip closure has taken place, sometimes producing a notch in it or a bifid uvula.

The way in which cleft lip and palate develop has been clarified considerably in recent years as the morphogenetic movements of the involved tissues have been better understood. Three recent findings worth a brief comment are:

- Primary palate is formed by the fusion of the lateral nasal process with the medial nasal process. Forward movement of the lateral nasal process during formation of primary palate keeps it in contact with medial nasal process. Interference with this movement can lead to clefting of the palate. Maternal smoking has been shown to be a major factor in the etiology of cleft lip and palate. The mechanism is thought to be hypoxia induced failure of the movement of the lateral nasal process. It interferes with oxidative phosphorylation of the cells, thereby reducing the synthesis of ATP which supplies energy for the morphogenetic movements.
- A genetic predisposition has also been found. Alterations in the genetic code for TGF α , NADH dehydrogenase were found to be associated with cleft lip and palate.
- Closure of the secondary palate depends on removal of the tongue from between the palatal shelves. A relatively large tongue in the affected twin of a monozygotic pair discordant for cleft palate seems to be a frequent finding. It is now clear that almost all cases of isolated cleft palate are related to problems in tongue removal, shelf elevation and contact of the shelves at the proper time.

Syndromic cleft lip: Two of the commonest syndromes associated with cleft lip are Vander-Woude's syndrome and cleft lip with ectodermal dysplasia.

Achondroplasia

This is caused by the failure of primary growth cartilages of the limbs and cranial base to grow properly. It is transmitted as an autosomal dominant trait. Forward growth of the mid face is produced by the normal lengthening of the anterior cranial base, which in turn is dependent on the growth at sphenooccipital, intersphenoidal and spheno-ethmoidal synchondroses. In achondroplasia, growth is diminished at these synchondroses. The resultant features include short arms, legs and characteristic midface deficiency (most accentuated at the bridge of the nose). The anterior cranial base is of normal length and the posterior cranial base length is shorter.

Premature Closure of Cranial and Facial Sutures

Premature fusion of the midsagittal or posterior cranial sutures can produce deformities of head without affecting the face, e.g. Scaphocephaly—premature closure of sagittal suture. Unilateral fusion along the coronal suture ring (plagiocephaly) has the potential to produce facial as well as cranial asymmetry. Premature fusion of sutures may cause secondary problems in the cranial base.

Crouzon's syndrome results from the premature fusion of the posterior and superior sutures of the maxilla along the walls of the orbit with cranial base involvement. It is characterized by symmetric maxillary deficiency that affects the infraorbital area. It is also characterized by shallow orbits resulting in protruding eye balls. The fusion extends to the cranium. Three fourths of the patients have fusion of the coronal, sagittal and or lambdoidal sutures.

Apert's syndrome is characterized by fusion of multiple facial and cranial sutures and early fusion of the synchondroses of the cranial base. These patients have an appearance similar to Crouzon's syndrome and have syndactyly as an additional clinical feature. Another important feature is that the metopic suture and anterior fontanelle are characteristically open at birth and during infancy in these patients, leading to pronounced frontal bossing and a high steep forehead.

ABNORMAL GROWTH PATTERNS

It is appropriate in the present context to review those conditions of abnormal facial structure which may have been caused, either wholly or in part, by abnormality

of growth mechanisms or growth patterns. It is well recognized that facial abnormality is a feature of many pathologic conditions and that there are different causes. Enlow et al have classified them into two broad categories with respect to the time of their recognition and are commonly used: (1) Abnormality of early embryonic development (2) Abnormality of late fetal or postnatal growth.

In Group 1, the early differentiation of tissues and developmental processes would be affected. Examples would include conditions of hemifacial microsomia, micrognathia, Apert syndrome, Crouzon syndrome, and cleft lip and palate. In each of these conditions some modification of later growth patterns may be observed as a consequence of the developmental abnormality.

In Group 2, it is presumed that the abnormal process becomes operative after the embryonic period, that is, after about the third month of fetal life. This group would include abnormalities due to trauma, endocrine malfunction, iatrogenic aspects of cleft lip and palate conditions, and skeletal malocclusions.

Group 1 abnormalities: It is noteworthy that the majority of craniofacial abnormalities of clinical interest exist within a normal or potentially normal tissue environment. Hemifacial microsomia frequently manifests as partial agenesis of the mandible in association with skin and auricular anomalies. Surgical correction of the mandibular defects frequently fails because the bone grafts are resorbed without replacement by new bone. A new approach to this problem, which appears to facilitate bone development in the graft matrix, has been deduced from studies of the relationship between function and morphology. The method involves preparation of the surgical site so that the space desired for the graft is precisely determined and maintained during the healing process. This maintenance can be obtained through use of an interocclusal appliance. Congenital micrognathia of the mandible, especially when associated with cleft palate, suggests a hypoplasia of the mandibular arch (Meckel's) cartilage in the embryo. Mandibular growth may subsequently compensate for a large part of the earlier retrognathia. Therefore, research for methods to accelerate elongation of the mandible to the point of relieving the airway in the critical weeks after birth, merits consideration.

The Apert and Crouzon syndromes are characterized by failure of midfacial growth with a tower-shaped cranial

vault and frontal protrusion. These abnormalities are attributed to premature synostoses of various sutures of the cranial base, vault, and maxillae. There may be a subsequent complication of elevated intracranial pressure in Apert syndrome. The syndactyly in the case of Apert syndrome suggests a mesenchymal response failure during the 33- to 39-day period of ovulation age. Since the entire nasal region is greatly reduced, an abnormality of the cranial base and nasal capsule in the embryonic chondrocranium may be involved.

Exploration of the retromaxillary region during surgical treatment of Apert syndrome has indicated a sphenomaxillary synostosis. Very little is known of the pathogenesis of this craniofacial condition, and no histologic study of the involved structures has been carried out. Knowledge relating to time of onset and time of suture synostosis obtained during surgical intervention, is needed. Anatomic research could shed light on whether maxillary hypoplasia is due to synostoses of the sutures or, perhaps, due to failure of normal differentiation in the anterior chondrocranial structures.

Synostoses of cranial sutures are known to relate to static growth relationships. It is possible that orthopedic treatment of these patients might be beneficial, provided the synostosis has not already occurred. Early surgical reshaping of the cranial vault by cranioplastic techniques still leaves the problem of the depressed middle third of the face.

Cleft lip and palate conditions give rise to secondary deformities of the middle third of the face during the embryonic and early fetal period. These are well established by the twelfth fetal week and progress only slowly thereafter. At birth they appear to be relatively stable entities in which basic growth mechanisms may be substantially normal. The problem, then, in the absence of severe tissue deficiency, is the presence of a deformity: structural displacement, malformation, and underdevelopment of the maxillae.

From a clinical viewpoint, it has been repeatedly asked: "If left untreated, would the middle third of the face attain normal size?" The evidence suggests that under such conditions the maxillae do grow to an adequate size. Nevertheless, it is evident that the functional impairment of persisting clefts might deprive the individual of full growth expression. The question still remains: "Does failure of normal function in patients with oral clefts significantly reduce growth of the upper jaw?"

Iatrogenic interference with growth is currently blamed for much of the abnormality of the facial profile in the adult oral cleft patient. In those cases of bilateral or unilateral complete clefts of the lip and palate, there is little doubt that a combination of maxillary arch collapse and growth inhibition contributes to the resultant deformity.

Research to define the nature and causes of the cleft lip and palate deformities (namely, the displacement deformity, tissue deficiency, and growth inhibition) in the context of normal facial growth mechanisms is still needed.

Group 2 abnormalities: A high percentage of those patients requiring orthodontic treatment show an imbalance in the relative size of the upper and lower jaws. In some cases, this pattern can be established before birth in relation to the cartilaginous components of the face, namely, the nasal capsule, the cranial base, and the mandibular arch cartilage. In other cases, the deviant pattern arises later, largely the product of postnatal growth processes. A good deal of our uncertainty in the diagnosis and prognosis of unacceptable facial relationships relates to our lack of knowledge of their natural history. There has been a tendency to abstain from corrective measures until the growing period has ended, when adaptation of regional organs to the abnormality is complete and the options for treatment are reduced. If an undesirable relationship is established early and persists, correctional measures could be appropriate at any time during the growing period.

On the basis of the biologic, interdisciplinary approach which constitutes "orthocephalics," the optimal rationale for diagnosis and treatment planning in severe craniofacial skeletal anomalies will have at least three phases or goals of treatment:

1. The earliest possible diagnosis and elimination of the pathologic process responsible for the skeletal anomaly. The main objective at this time is to focus on identification of the pathologic stimulus. Early interception of the pathognomonic process which is a way of restoring the capacity for subsequent growth at that site is the first goal.
2. The interception and reversal of secondary skeletal deformities by means of force-induced articular and skeletal remodeling. The objective at this stage is to focus on the spectrum of skeletal deformities that constitute biologic adaptations to the pathologic defect. The therapeutic forces should be applied directly to the skeletal sites that require remodeling in order to achieve a functional balance.
3. Supplementation of previous treatment by means of minor plastic and reconstructive surgery aimed at cosmetic rather than functional correction.

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Chapter 16

Growth Considerations in Stability of Orthodontic Treatment

CHAPTER OUTLINE

- **Changes Related to Growth**
 - Growth Changes in Forward Rotating Mandible
 - Growth Changes in Posterior Rotation of the Mandible
 - Maxillary Growth Rotation and Stability
 - Changes in Arch Width and Arch Length
 - Growth Considerations in Stability of Extraction and Non-extraction Treatment
 - Growth Considerations in Retention Period after Treatment in Various Types of Malocclusion
 - Retention after Class II Correction
 - Retention after Class III Malocclusion

Retention is defined as the process of holding teeth in their optimal esthetic and functional position long enough to aid in their stabilization. Success in orthodontic treatment is achieved not only by correct diagnosis, logical treatment planning and accurate treatment timing, but also by planning of retention. The results achieved after active orthodontic treatment are maintained by retention appliances to prevent relapse. One of the main problems in orthodontics is failure to maintain the corrected relationships. The improvement achieved by long duration treatment is lost in varying degrees after the removal of the retention appliance. Studies on long term assessment of post treatment results have proved that relapse occurs in most cases. There has always been a question about the achievement of long-term stability.

Relapse of corrected position of teeth after successful orthodontic treatment is a source of annoyance to the orthodontist. Nanda has stated that the change related to growth, maturation, and aging of the dentition and occlusion is one of the important reasons for the instability of occlusion following orthodontic treatment. Relapse

is mainly due to the skeletal changes as a result of the patient's growth pattern. Despite considerations being given to skeletal relationships at the initiation of and during the orthodontic treatment, very meagre consideration is given to the skeletal relationship during retention. There are two reasons for this:

1. It is assumed that skeletal supervision is considered secondary to dental relationship during orthodontic treatment. Importance is given to the proper interdigitation of the posterior teeth.
2. It is assumed that nothing can be done to control the growth pattern of the patient.

The truth is that most of the patients whose orthodontic treatment is completed are still going through their pubertal growth spurt, which is more important in boys than in girls, as they mature at a later period. The failure to recognize the effects of dentofacial growth after orthodontic treatment and its effect on the morphology of the jaws might have an unfavorable effect on the stability of orthodontic treatment. Therefore, retention appliances should be selected based on the dentofacial morphology and the expected magnitude and direction of growth.

CHANGES RELATED TO GROWTH

Facial growth does not generally stop with puberty, and the growth of the jaws continues till adulthood. This progressive growth of the face results in a less convex face, a less protrusive dentition with more upright incisors and a more prognathic mandible. The amount of changes produced varies both in males and females. In males, these effects appear later, continue longer and produce more marked changes. In a study by Bishara

about the changes in the face in adulthood, he concluded that in male and female subjects, skeletal anteroposterior and vertical linear dimensions continue to change between 26 and 46 years of age.

In both male and female subjects, the lips became more retruded relative to the nose and chin. The implication is that orthodontic treatment at earlier ages should not result in an overly straight soft tissue profile and overly retrusive lips, since the expected changes in the relative positions of the lips, nose, and chin may exaggerate these characteristics.

In both male and female subjects, interincisor and intercanine arch widths decreased, total arch lengths decreased, and anterior crowding increased. Clearly, these findings have important clinical implications regarding the long range stability and retention of the treatment results.

Generally, there is high variability in facial growth. This was demonstrated by the Bjork implant studies. The variation is seen in the direction of facial growth as well as in the growth of maxilla and mandible, and in the eruption of teeth in the jaws. Bjork, from his implant study for mandibular growth, concluded that the range of variation of condylar growth in untreated normal subjects may be as much as 42° , with a slight upward and forward growth direction being most common, while some people showed posterior condylar growth which eventually showed distinct variations in the eruption of teeth.

Growth Changes in Forward Rotating Mandible

- In a pronounced forwardly rotating mandible, the mandible teeth erupt and migrate mesially.
- The lower incisors are prevented from moving forward—thereby increasing the crowding in the lower arch and producing deep bite.
- For an anterior rotating mandible with a stable occlusion, the fulcrum is at the incisors.

The goal of orthodontic treatment is to establish and maintain normal, overjet and overbite relationships by creating a solid fulcrum point at the incisors. By positioning so that the interincisal angle is not too obtuse and the lower incisors are not too upright, with the required amount of torque in the upper incisors, the anterior occlusion will be stable in an upward and forward rotating mandible. Keeping the lower incisors too upright

in forward rotating mandible will cause further uprighting of the incisors and eventually crowding will be the result.

In patients with severe class II malocclusions and deep bite wherein treatment is completed early, the lower incisors stability poses a serious problem. A fixed lingual retainer banded to the second deciduous molar or first permanent molar and a passive bite plane along with a functional appliance worn at night could be given as retentive devices. Although the continued facial growth late in adolescence is outside the practitioner's control, it is an important contributor to the stability of the treatment results. In particular, the residual forward growth of the mandible accommodates and largely masks the tendency of the maxilla to grow forward and the crown of the upper molar to drift mesially within the bone.

Growth Changes in Posterior Rotation of the Mandible

In posterior condylar rotation, the amount of vertical condylar growth determines the amount of increase in posterior facial height. This type of rotation is less common and here, the amount of increase in anterior facial height exceeds that of the posterior facial height. As the direction of eruption of incisors is more vertical, the tendency towards retroclination and late incisor crowding is also increased. Hence, long-term stabilization of lower anterior teeth is absolutely necessary.

Maxillary Growth Rotation and Stability

Maxillary growth rotation is of less intensity than the mandible. Due to the rotation of the maxilla, similar to the mandible, the posterior teeth migrate mesially and the anteriors show less forward movement, and hence, more chance of incisor crowding. The lower lip plays a major role in the development of upper incisor crowding. Studies by Thuer have proved that the upper lip is hypotonic in class II division 2 malocclusion and that the lower lip is responsible for the upper crowding. The same mechanism may be responsible for the relapse of maxillary anterior crowding after treatment.

In patients with pronounced forward growth of the jaws, there is more tendency towards class II molar relation, due to more mesial inclination of upper molars and distal inclination of mandibular molars due to growth rotation. These changes are more or less pronounced, depending on the intercuspitation and the function of the soft tissue matrix.

Changes in Arch Width and Arch Length

Arch width and arch length changes with time. In a study by Bishara about the changes in the arch width from birth up to 45 years of age, he concluded the following:

- Between 6 weeks and 2 years of age, i.e. before the complete eruption of the deciduous dentition, there was significant increase in the maxillary and mandibular anterior and posterior arch width in both boys and girls.
- Inter canine and intermolar width significantly increased between 3 and 13 years of age in both the maxillary and mandibular arches. After the complete eruption of the permanent dentition, there was a slight decrease in the dental arch width, more in the inter canine than in the intermolar width.
- Mandibular inter canine width, on the average, was established by eight years of age, i.e. after the eruption of the four incisors. After the eruption of the permanent dentition, the clinician should either expect no change or a minimal decrease in arch width.

Changes in arch width continue even after the cessation of growth. The changes in the arch width are variable. There is a tendency towards decrease in the arch width and length in early adulthood which is active between the 20 to 30 year age span. Beyond 30 years of age, the process continues and the constrictive trend is very minimal. These unfavorable changes in arch width and arch length result in crowding. The changes occur regardless of whether the patient had orthodontic treatment or not. Reidel has reported that even patients who were at least five years out of retention returned to their original dimensions. Walter has reported that slight increase in inter canine width can be maintained after an adequate period of retention. This adequate period was given as five years by Arnold.

Changes in post-treatment inter canine width were also seen in patients, who had undergone extractions. In Shapiro's study, he concluded that changes in inter canine width were more stable in cases with class II division 2 malocclusion and that the arch length reduction in cases with class II division 2 was significantly less than in cases with class I and II division 1 malocclusion.

Luiz G Gandini Jr and Peter H Buschang found that the width of maxillary and mandibular basal structures increased during late adolescence. Changes in the maxillary width explained approximately 50 percent of the variation among subjects with mandibular width

changes. Maxillary width increased more than mandibular width, and the width changes were related with the subjects' growth potential. Subjects with the greatest growth potential showed the greatest width changes and might be expected to tolerate the greatest amount of therapeutic expansion.

Growth Considerations in Stability of Extraction and Non-extraction Treatment

In patients with anterior growth rotation, extraction of teeth in lower arch should be avoided and the potential for sagittal and transverse expansion should be considered before deciding on extraction. As a result of extraction, an unstable occlusion is created and the front teeth are too upright with an obtuse interincisal angle which lacks an anterior fulcrum point. If extractions are carried out, they should be done very early before the growth spurt or before the growth pattern is clearly expressed. Retention is more critical in these extraction cases and must be maintained until the growth of the condyles are completed.

When the condylar growth is directed posteriorly, the natural tendency of the mandibular incisors to become more crowded with time continues throughout the growth period. Therefore, extraction decisions are not made too early and in most instances, where posterior rotation is anticipated, the extraction decision is delayed until the completion of pubertal growth spurt. The degree of growth rotation and the natural tooth migration is unpredictable in this growth pattern. Hence, a permanent lower lingual bonded retainer is recommended.

Growth Considerations in Retention Period after Treatment in Various Types of Malocclusion

Short Face Syndrome

As vertical growth occurs several years after the pubertal growth spurt, the individuals with short face syndrome with deep bite require a maxillary removable retainer with a bite plane for several years after fixed appliance orthodontics. Failure to recognize the dominant horizontal pattern of growth of the individual may result in a "dished in face" with or without extraction. Additional growth of soft tissues especially the nose would further increase the concavity of the face.

Long Face Syndrome

A high pull face bow is required to hold the molar position vertically and prevent further dentoalveolar growth and worsening of the facial profile.

Retention after open bite correction: Control of erupting molars is the key to retention in open bite patients. An alternative to high pull head gear is a posterior bite block that provides several millimeters of jaw separation, e.g. open activator or bionator. The bite block produces the stretch of the soft tissues thus preventing the eruption of the molars.

Patients with severe open bite problems can be given conventional maxillary and mandibular retainers for daytime wear and an open bite bionator as a night time retainer, from the beginning of the retention period.

Jean Driscoll-Gilliland et al, in their study on stability of treatment due to growth, concluded the following:

- Significant growth, especially in the posterior and lower anterior facial height, occurred beyond the age when orthodontic treatment is typically completed.
- Tooth Size Arch Length Discrepancy (TSALD) increased more in the untreated subjects than in the treated subjects, and the difference might be a result of the combined effects of growth and treatment.
- The subjects who had greater growth in the vertical dimension and lower incisor eruption had larger increase in space irregularity.

Retention in Class II Correction

Relapse in Class II correction can occur as two processes: (a) Short-term relapse occurring due to local periodontal and gingival factors. (b) Long-term relapse due to altered jaw position.

The short-term relapse can be controlled by over-correction and by avoiding too much proclination of lower incisors. The long-term relapse occurs due to differential jaw growth. This can be controlled in two ways:

1. After the fixed appliance therapy, head gear is given on a part time basis to control the molar position. Compliance is the major problem with head gear. However, patients who were wearing the head gear during treatment show good cooperation in wearing it during retention phase also.
2. A functional appliance (activator or bionator) to retain the occlusal relationship and tooth position. The patient is advised to wear the appliance on a part

time basis during the night and the conventional retainers during the day time. The wear of functional appliance is good for people with skeletal discrepancy.

Retention after Class III Malocclusion

Retention after class III correction is difficult due to the continuous growth of the mandible. The chin cup, as a restraining force to the mandible, is ineffective and only causes downward and backward rotation of the mandible leading to an increase in the vertical facial height. Surgical treatment after growth completion is the only option for patients with vertical growth pattern and class III malocclusion. A functional appliance or a tooth positioner is usually sufficient for a mild class III malocclusion.

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Chapter

17

Temporomandibular Joint

CHAPTER OUTLINE

- Anatomy
- Embryology
- Histology
- Origin and Evolution
- Condylar Growth and Glenoid Fossa Displacement During Growth and in Malocclusion

The temporomandibular joint (TMJ) is a cardinal feature that defines the class mammals and separates mammals from other vertebrates. The name of the joint is derived from the two bones which form the joint:

- The upper temporal bone which is part of the cranium (skull).
- The lower jaw bone called the mandible.

Temporomandibular joint is one of the most complex joints in the body and is the area in which the mandible articulates with the cranium. It is a highly specialized synovial joint characterized by many unique features:

- One of the unique features of the temporomandibular joint is the *articular disk*. The disk is composed of fibrocartilaginous tissue which is positioned between the two bones that form the joint. The TMJ is one of the two synovial joints in the human body with an articular disk, another being the sternoclavicular joint. The disk divides each joint into two. The lower joint compartment formed by the mandible and the articular disk is involved in rotational movement (opening and closing movements). The upper joint compartment formed by the articular disk and the temporal bone is involved in translational movements (sliding the lower jaw forward or side to side). The inferior compartment allows for the pure rotation of

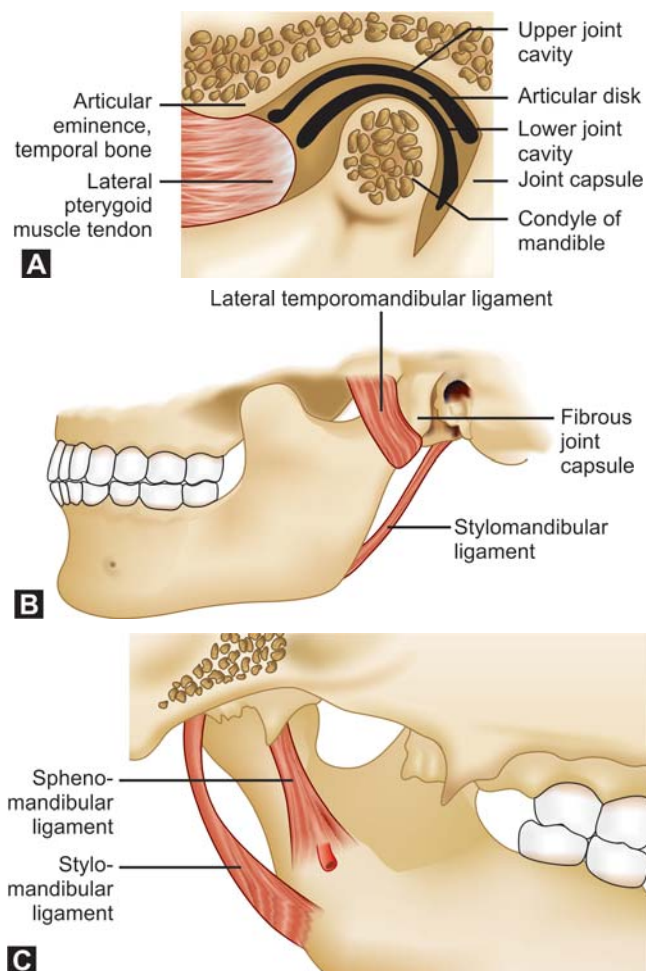
the condylar head, which corresponds to the first 20 mm or so of the opening of the mouth. Beyond 20 mm of opening, the mouth can no longer open without the superior compartment of the TMJ becoming active. At this point, if the mouth continues to open, not only is the condylar head rotating within the lower compartment of the TMJ; but the entire apparatus (condylar head and articular disk) translates, or slides forward in the glenoid fossa and down the articular eminence of the temporal bone; thus incorporating an anterior movement into the further opening of the mouth. This can be demonstrated by placing a resistance fist against the chin and trying to open the mouth more than 20 mm.

- *A compound joint:* The TMJ is formed by the mandibular condyle fitting into the mandibular fossa of the temporal bone. Separating these two bones from direct articulation is the articular disk. By definition, a compound joint requires the presence of at least three bones, yet TMJ is made of only two bones. Functionally, the articular disk serves as a nonossified bone that permits the complex movements of the joint. As the articular disk functions as a third bone, the craniomandibular articulation is considered to be a compound joint.
- *Ginglymoarthrodial joint:* *Ginglymus*-hinge joint; *arthrodial* joint—gliding joint, is a synovial joint which admits only gliding movement. The TMJ is a ginglymoarthrodial joint, referring to its dual compartment structure and function (ginglymo and arthrodial). The condyle articulates with the temporal bone in the mandibular fossa. The mandibular fossa is a concave depression in the squamous portion of the temporal bone.

- The joints, although anatomically distinct, one on either side, function in unison. Independent movements are restricted as the two sides are coupled.
- This joint develops very late. At 45 to 48 mm stage of embryo when all other joints are fully formed, this joint is incomplete. In 320 mm stage, excepting the articular eminence, the rest of the joint is formed. Only by the 12th year of life the joint is fully developed in all respects.
- This joint is not designed for bearing weight.
- Movements are not only guided by shape of bones, muscles and ligaments but also, by the occlusion of teeth.

ANATOMY OF TEMPOROMANDIBULAR JOINT

There are six main anatomical components of the TMJ (Figs 17.1A to C):



Figs 17.1A to C: TMJ and its components: A—Articular surfaces, capsule and articular disk; B—Major ligaments; C—Minor ligaments

1. Articular surface of mandibular condyles
2. Articular surface of the temporal bone
3. Capsule
4. Articular disk
5. Ligaments
6. Lateral pterygoid.

Articular Surfaces

- **Mandibular component:** It consists of an ovoid condylar process seated atop a narrow mandibular neck. It is 15-20 mm side to side and 8-10 mm from front to back with its long axis being at right angles to plane of ramus. Thus it does not lie in the frontal plane of skull as the two sides of mandible spread wide posteriorly. Thus, if the long axes of the two condyles are extended medially, then they meet approximately at the basion on the anterior limit of foramen magnum. This forms an angle which opens towards the front, varying from 145 to 160 degrees. The lateral pole of the condyle is rough, bluntly pointed and projects only moderately from the plane of ramus while the medial pole extends strongly inwards from the plane of the ramus. The articular surface lies on its anterosuperior aspect, thus facing the posterior slope of articular eminence of temporal bone. It continues medially further down and around the medial pole of condyle and faces into glenoid process of temporal bone when the jaw is held in occluded position.
- **Cranial component:** The articular surface of the temporal bone or facies articularis is more complicated than that of the mandible. It is situated on the inferior aspect of temporal squama, anterior to tympanic plate. The parts of the articular surface of temporal bone are:
 - **Articular eminence:** It is the entire transverse bony bar that forms the anterior root of the zygoma. This articular surface is most heavily travelled by the condyle and the disk as they ride forwards and backwards in normal jaw function.
 - **Articular tubercle:** It is a small rough bony knob raised on the outer end of articular eminence. It projects below the level of articular surface and serves as attachment of the lateral collateral ligament of joint. Though termed as the articular tubercle, it is non-articulating.
 - **Preglenoid plane:** It is the slightly hollowed, almost horizontal articular surface continuing anteriorly from the height of eminence.

- *Posterior articular ridge (lip) and postglenoid process*: The tympanosquamosal suture is divided by the protruding inferior edge of the tegmen tympani into an anterior petrosquamosal and a posterior petrotympanic fissure. The posterior part of mandibular fossa, i.e. posterior border of squamous temporal (or anterior margin of petrosquamous suture) is elevated to form a ridge known as the posterior articular ridge or lip. This ridge increases in height laterally to form a thickened cone shaped prominence called the post-glenoid process immediately anterior to external acoustic meatus.
- The lateral border of mandibular fossa is usually raised to form a slight crest joining the articular tubercle in front with the postglenoid process behind.
- Medially, the fossa narrows considerably and is bounded by a bony wall, the entoglenoid process which passes slightly medially as the medial glenoid plane.

The roof of the mandibular fossa separating it from the middle cranial fossa is always thin and even in a heavy skull, translucent. This is clear evidence of the fact that the articular fossa, although containing the posterior rim of disk and the condyle, is not a functional stress-bearing part of the craniomandibular articulation. This function is normally always between the condyle and the disk on one hand and the disk and the articular eminence with its extended planes on the other.

Capsule

The capsule is a fibrous membrane that surrounds the joint and incorporates the articular eminence. It is attached to the articular eminence, the articular disk and the neck of the mandibular condyle.

Articular Disk

The articular disk is a fibrous extension of the capsule, shaped to accommodate the shape of the condyle and the concavity of the mandibular fossa. The disk functions as the articular surfaces against both the temporal bone and the condyles and divides the joint into upper and lower compartments. Its upper surface is concavo-convex from before backwards to fit with the articular eminence and the fossa of the temporal bone. Its lower surface

is concave to fit into the convex head of the mandible. The disk is thicker medially than laterally (Fig. 17.2).

In the sagittal plane (Fig. 17.3), the disk can be divided into three regions according to thickness, the anterior portion, intermediate, and the posterior zone. The disk is thinnest at the central intermediate zone and becomes considerably thicker both in anterior and posterior zones. The disk is attached posteriorly to a region of loose connective tissue that is highly vascularised and innervated known as retrodiskal tissue.

Retrodiskal lamina: The superior retrodiskal lamina attaches to the articular disk and posteriorly to the

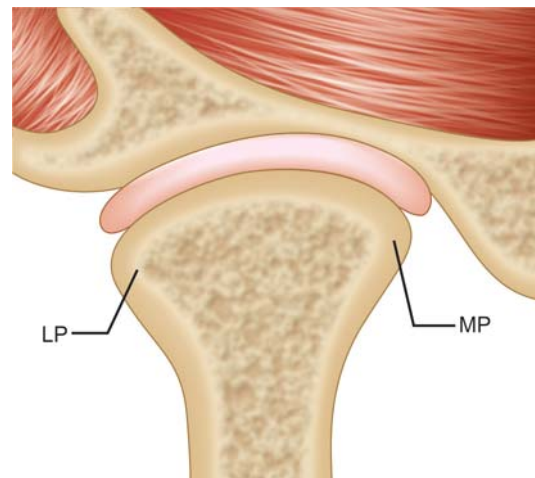


Fig. 17.2: Articular disk interposed between fossa and condyle. Disk is thicker medially than laterally. LP—Lateral pole; MP—Medial pole

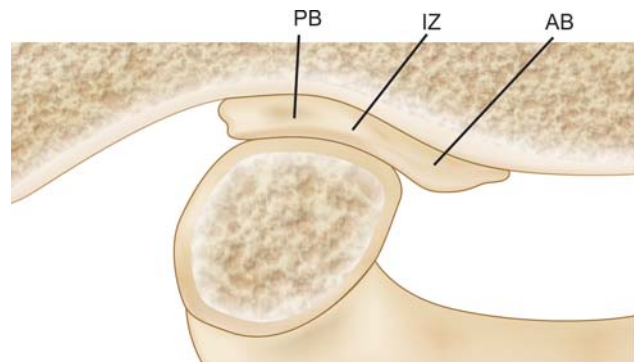


Fig. 17.3: View of the articular disk in sagittal plane. Condyle is located in the thinner intermediate zone. Posterior border is the thickest and anterior border is thicker than intermediate zone. PB—Posterior border; IZ—Intermediate zone; AB—Anterior border

tympanic plate. The inferior retrodiskal lamina is attached to the inferior border of the posterior edge of the disk and to the posterior margin of the articular surface of the condyle. The remaining portion of the retrodiskal tissue is attached to a large venous plexus, which fills with blood as the condyle moves forward. The retrodiskal tissue, unlike the disk itself, is vascular and innervated, and in some cases of anterior disk displacement, the pain felt during movement of the mandible is due to the condyle pressing on this area.

The superior and inferior attachments of the anterior region of the disk are to the capsular ligament, which surrounds most of the joints. The inferior attachment is to the anterior margin of the articular surface of the condyle. Anteriorly, between the attachments of capsular ligament, the disk is also attached by tendinous fibers to the lateral pterygoid muscle.

Ligaments

The ligaments of the joint are made up of collagenous connective tissue which has a particular length and does not stretch under normal conditions. If extensive force is applied to a ligament or if force is applied for a long duration, they do elongate compromising the function of the ligament, thereby altering the joint function. Ligaments do not actively enter into joint function. Rather, they act as passive restraining devices to limit

and restrict the border movements. However, the movements of the mandible made past the extent functionally allowed by the muscular attachments will result in painful stimuli, and thus, movements past these more limited borders are rarely achieved in normal function. The ligaments can be classified into major ligaments and minor ligaments (Fig. 17.4).

Major Ligaments

- Capsular ligament
- Temporomandibular ligament
- Collateral ligament.

Minor Ligaments

- Stylomandibular ligament
- Sphenomandibular ligament.

Capsular Ligament

The fibers of this ligament encompass the entire articular surface of the joint. It is superiorly attached to the temporal bone along the borders of the articular surface; anteriorly to articular eminence; and inferiorly to the neck of the condyle (Fig. 17.5).

Functions

- It resists any medial, lateral or inferior forces that tend to separate or dislocate the articular surface.
- It encompasses the joint, thus retaining the synovial fluid.
- This ligament is well innervated and provides proprioceptive feedback regarding the position and movement of the joint.

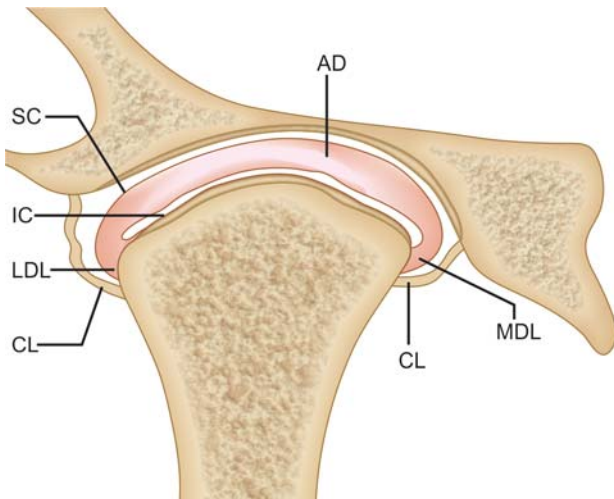


Fig. 17.4: Anterior view of temporomandibular joint. AD—articular disk; SC—superior joint cavity; IC—inferior joint cavity; LDL—lateral disk ligament; CL—capsular ligament; MDL—medial diskal ligament



Fig. 17.5: Lateral view of capsular ligament. It extends anterior to the articular eminence and covers the whole articular surface of the joint

Temporomandibular Ligament

It is the thickened lateral portion of the capsular ligament, and has two parts: the outer oblique portion (OOP) and inner horizontal portion (IHP) (Fig. 17.6). The outer oblique portion extends from the outer surface of the articular tubercle and the zygomatic process posteroinferiorly to the outer surface of the condyle. The function of the outer oblique portion is to limit the rotational opening movements. The inner horizontal portion extends from the outer surface of the articular tubercle and zygomatic process posteriorly and horizontally, to the lateral pole of the condyle and the posterior part of the articular disk. Its function is resisting the posterior movement of the condyle and disk, thus protecting the retrodiskal tissues from trauma due to posterior displacement.

Collateral (Diskal) Ligament

It consists of medial diskal ligament and lateral diskal ligament. Medial diskal ligament is attached to the medial pole of the condyle, to the medial edge of the disk and the lateral diskal ligament attaches the lateral edge of the disk to the lateral pole of the condyle. Diskal ligaments divide the joint mediolaterally into superior and inferior joint cavities. The diskal ligament restricts the movement of the disk away from the condyle and allows the disk to move passively with the condyle as it glides anteriorly and posteriorly. Thus, they are responsible for the hinge movement of the TMJ (Fig. 17.4).

Minor Ligaments

These are accessory ligaments and are not directly attached to any part of the joint. The *stylomandibular*

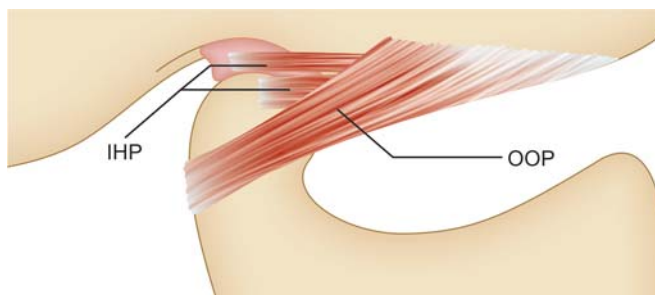


Fig. 17.6: Lateral view of temporomandibular ligament. Outer oblique (OOP) and inner horizontal (IHP) parts are the two distinct parts

ligament separates the infratemporal region (anterior) from the parotid region (posterior), and runs from the styloid process to the angle of the mandible. It limits excessive protrusive movements of the mandible. The *sphenomandibular ligament* runs from the spine of the sphenoid bone to the lingula of the mandible. It does not have any significant action in limiting mandibular movement (Fig. 17.7).

Nerve Supply and Blood Supply

Sensory innervation of the temporomandibular joint is derived from the auriculotemporal and masseteric branches of the fifth cranial nerve (otherwise known as the mandibular branch of the trigeminal nerve). The specific mechanics of proprioception in the temporomandibular joint involve four receptors: *Ruffini endings* function as static mechanoreceptors which position the mandible; *Pacinian corpuscles* are dynamic mechanoreceptors which accelerate movement during reflexes; *Golgi tendon organs* function as static mechanoreceptors for the protection of ligaments around the temporomandibular joint. Free nerve endings are the pain receptors for the protection of the temporomandibular joint itself.

Its arterial blood supply is provided by the branches of the external carotid artery, predominantly the superficial temporal branch. Other branches of the external carotid artery namely: the deep auricular artery, anterior tympanic artery, ascending pharyngeal artery,

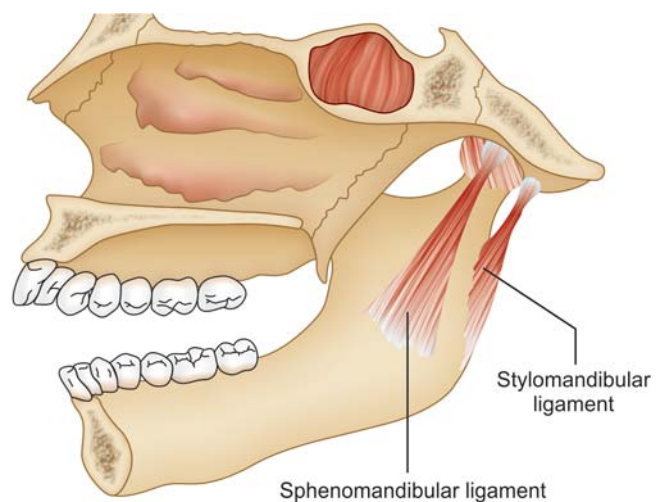


Fig. 17.7: Minor or accessory ligaments of temporomandibular joint

and the maxillary artery may also contribute to the arterial blood supply of the joint.

In order to work properly, there is neither innervation nor vascularization within the central portion of the articular disk. Had there been any nerve fibers or blood vessels, people would bleed whenever they move their jaws; moreover, the movement itself would have been too painful.

EMBRYOLOGY

The early TMJ structures emerged progressively from a block of embryonic mesenchymal cells interposed between the developing temporal bone and mandible. This early block of mesenchymal tissue is a “developmental field” whose normal morphogenesis into discrete anatomic parts can be interfered with, in some significant and timely manner resulting in the anomalous development of one or more structures evolving from that “developmental field”.

The critical period in the early prenatal morphogenesis of the human temporomandibular joint is approximately during the time of early 7 to 11 weeks of fertilization age. There appears to be a definite pattern or sequence in the early shaping of each component of the temporomandibular joint relative to structure and chronology. The chronological events leading to development of the temporomandibular joint is enumerated in Table 17.1.

Table 17.1: Chronology of TMJ development

Timing	Structures developed
Approximately 7-7.5 weeks	First appearance of the temporal bone articular fossa.
Approximately 7-7.5 weeks	Beginning signs of mesenchymal condensation and shaping of the mandibular condyle.
32-34 days	First signs in the development of the lateral pterygoid muscle.
7-7.5 weeks	First appearance of the articular disk.
9-11 weeks	Articular capsule development initiated.
17 weeks	Articular disc fully formed.
10 weeks	Initiation of lower joint cavity.
11.5 weeks	Initiation of upper joint cavity.
17.5 weeks	Fully formed joint cavities.

Primary Joint

At about 7th week of intrauterine life, the Meckel's cartilage extends from the midline backward and dorsally, acting as a scaffold to the developing mandible. It terminates at the malleus (or in cephalometric terminology, the articulare) and articulates with the incal cartilage (the quadrate in nonmammals) forming the primary joint and any movement of the early jaws occur between them. Embryonic and fetal movements during the course of human development are essential for joint formation. It is stated that the factors promoting joint formation are intrinsic and genetically determined. This primary joint exists for about four months until the cartilages ossify and become incorporated in the middle ear.

Secondary Joint

Two distinct regions of mesenchymal condensation (blastemata) appear at three months of gestation:

1. Temporal blastemata
2. Condylar blastemata.

Temporal Blastema

Temporal blastema gives rise to articular eminence and glenoid fossa. The first appearance of the temporal bone articular fossa occurs at approximately 7 to 7.5 weeks (21 mm crown rump length [CRL]) as a visible condensation of the deeper staining stellate cells comprising the embryonic mesenchyme (temporal blastema). Spicules of primary cancellous bone appear most prominently at 10 to 11 weeks (60 mm CRL). The shape of the articular fossa is initially convex during the first weeks of its development up to 9 weeks (40 mm CRL). After that time, the fossa progressively takes on its definitive concave shape, which matches the shape of the condylar head.

Condylar Blastema

The condensation and shaping of the mandibular condyle occur at about the same time as for the articular fossa. This mass is superiorly convex. Condylar cartilage cells first appear between 9 and 10 weeks (40 to 50 mm CRL). The shape of this early cartilaginous condyle is that of the mesenchymal mass from which it arises. With few exceptions, it is convex. From this time onwards till the 10th week, the ossification of the condyle and articular

Table 17.2: Stages of temporomandibular development

Stages	Events
Blastematic stage (weeks 7-8 of development)	Corresponds with the onset of the organization of the condyle and the articular disc and capsule. During week 8, intramembranous ossification of the temporal squamous bone begins.
Cavitation stage (weeks 9-11 of development)	Corresponding to the initial formation of the inferior joint cavity (week 9) and the start condylar chondrogenesis. Week 11 marks the initiation of organization of the superior joint cavity.
Maturation stage	After week 12 of development.

fossa are synchronous. When a difference in ossification is observed, it is the temporal bone that is more advanced. After the 10th week, the continuing ossification of the articular fossa appears more advanced in terms of increased cortical thickness and density of bony trabeculae. Up to about 10 weeks, the ossifying masses of the articular fossa and mandibular condyle are separated by a continuous and unseparated block of dense staining mesenchyme.

The upper and lower joint cavities: They progressively appear as a group of small spaces or clefts within the mesenchymal tissue block that had earlier given rise to the articular fossa, disk, and condyle. Initiation of both the joint cavities is not synchronous. Small, coalescing clefts for the lower joint cavity appear at about 10 weeks (50 to 58 mm CRL), whereas those for the upper cavity are first seen at about 11.5 weeks (60 to 70 mm CRL). These small spaces or clefts between the mesenchymal cells in the area gradually enlarge and coalesce into larger spaces or cavities superior and inferior to the disk.

The three phases in the development of the TMJ are given in Table 17.2.

The TMJ provides the essential functional connection between the cranium and the upper and lower jaw. However, the primary function of the TMJ in general, and that of the mandibular condyle in particular, is not so simple, and it changes during development. The primary roles of the mandibular condyle are twofold, and they may be considered within three arbitrary phases of development. The first primary role of the mandibular condyle is directed toward growth, which is most evident and important prenatally and early postnatally (phase I), and diminishes in expression as development proceeds postnatally through adolescence (phase II). The second role of the mandibular condyle is directed towards mandibular articulation and its load-bearing capabilities.

Beginning in incipient form, as the mandible may move prenatally, the articular function gains primacy as the growth function diminishes during phase I. With adulthood (phase III), condylar growth has essentially ceased; though remodeling may continue throughout life, while articular function continues.

HISTOLOGY (FIG. 17.8)

Condyle of the Mandible

This is composed of cancellous bone covered by a thin layer of compact bone. The trabeculae radiate from the neck of the mandible and reach the cortex at right angles, giving maximal strength to the condyle. As the age progresses, the trabeculae thicken, decreasing the size of large marrow spaces. Red marrow in the condyle is of myeloid or cellular type which is replaced by fatty marrow in older individuals.

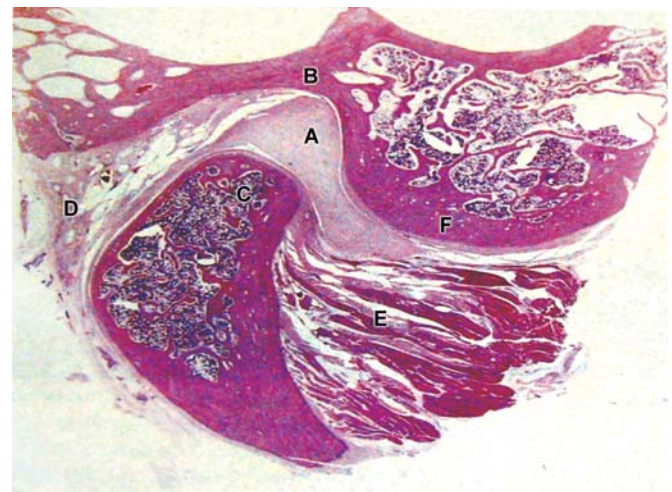


Fig. 17.8: Arrangement of tissues in TMJ. A— Articular disk; B— mandibular or glenoid fossa of temporal bone; C— condyle of mandible; D— capsule of joint; E— lateral pterygoid muscle; F— articular eminence

Roof of Glenoid Fossa

It consists of a thin compact layer of bone. The articular eminence is composed of spongy bone covered with a thin layer of compact bone. Areas of chondroid bone are commonly seen in the articular eminence, and in rare cases, islands of hyaline cartilage.

Articular Surfaces of the Mandibular Condyle and Fossa

They are lined by dense fibrous connective tissues which contain some elastic fibers. The articular layers are thicker over the convexity on the anterior part of the condyle and over the articular eminence of the temporal bone. It is composed of four distinct layers or zones (Fig. 17.9):

1. Articular
2. Proliferative
3. Fibrocartilagenous
4. Calcified cartilage.

Articular zone: It is the most superficial layer, found just adjacent to the joint cavity, forming the outermost functional surface. Unlike most other synovial joints, where the articular surfaces are covered by hyaline cartilage, the TMJ articulation is covered by a layer of fibrous tissue. This histological distinction reflects the joint's intramembranous development. It consists of tightly packed collagen bundles oriented parallel to the articular surfaces, allowing them to withstand the forces of movement. Fibrous cartilage affords several advantages

over the hyaline cartilage. It is less susceptible to the effects of aging, and therefore, less likely to breakdown and it has much better ability to repair than the hyaline cartilage.

Proliferative zone: It is mainly cellular, consisting of undifferentiated mesenchymal tissues, which are responsible for the proliferation of articular cartilage in response to the functional demands placed on the articular surfaces.

Fibrocartilagenous zone: This zone consists of the collagen fibrils arranged in bundles in a crossing pattern. The fibrocartilage appears in a random orientation, providing a three-dimensional network that offers resistance to the compressive and lateral forces.

Calcified cartilage: It is the fourth and the deepest zone. This zone is made up of chondroblasts and chondrocytes distributed throughout the articular cartilage. In this zone, chondrocytes become hypertrophic and die, causing their cytoplasm to be evacuated, forming bone cells from within the medullary cavity.

Articular Surface of Temporal Bone

The fibrous layer covering the articular fossa is thin in the articular fossa and thickens rapidly on the posterior slope of the articular eminence with two definite layers of fibrous tissue and a transitional zone in between them. The inner zone is characterized by the perpendicular arrangement of fibrous bundles to the bony surface while the outer zone runs parallel to the surface. Free surface of the fibrocartilage is devoid of any continuous cellular linings. Only isolated fibroblasts are situated on the surface of the fibrocartilage, characterized by the formation of long, flat cytoplasmic processes.

Articular Disk

The disk is a dense, collagenous, fibrous pad between the condylar heads and the articular surfaces. Elastic fibers are found only in relatively small numbers. The fibroblasts in the disk are elongated and send flat cytoplasmic processes into the interstices between the adjacent bundles. The disk is thinnest centrally and thickens in the periphery. Isolated clumps of more rounded, cartilage-like cells have also been described within the disk; the disk is therefore sometimes been called fibrocartilage. It is devoid of any blood vessels and nerves for the most

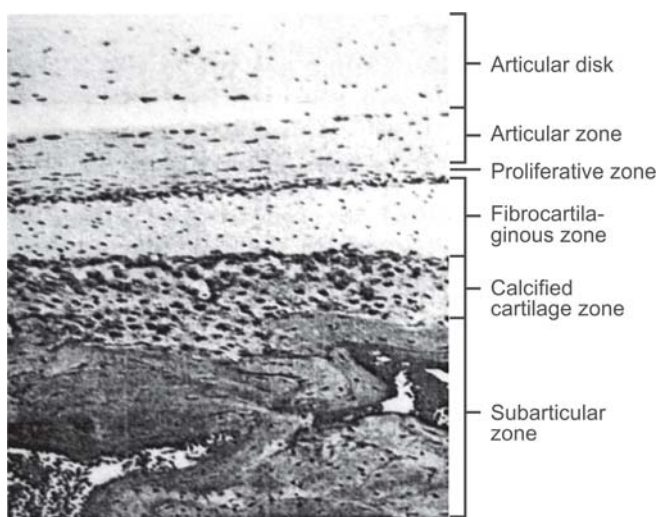


Fig. 17.9: Histological section of condyle showing the four zones

part, except for the extreme periphery of the disk which is slightly innervated.

The fibers in the disk comprise about 80 percent of type I collagen and 5 percent glycosaminoglycans of its dry weight. Of the 5 percent of glycosaminoglycans, 80 percent is chondroitin sulphate and about 15 percent is dermatan sulphate. In the thin central region of the disk, the collagen fibers are running mainly in an anteroposterior direction. In the thicker anterior and posterior regions of the disk, prominent fiber bundles run transversely with a mediolateral orientation. A very small amount of type III collagen has been described at the posterior attachment region of the disk. The localized area of fibrocartilage would be expected to contain a small amount of type II collagen.

Blood Vessels in the Posterior Part of the Articular Disk (Fig. 17.10)

The bulk of the disk is avascular and derives its nutrition by diffusion from the synovial fluid. The blood vessels are localized at the periphery. However posteriorly, in the bilaminar zone, the disk divides into superior and

inferior lamellae. The superior lamellae possess numerous blood vessels and elastin fibers. As the disk is pulled forwards by the lateral pterygoid muscle during jaw opening, the tissue of the superior lamella fills the space behind the migrating condyle. As the condyle moves backwards during the jaw closure, the disk returns to its original position aided by the elastic recoil of the superior lamella. The inferior lamella is relatively avascular and inelastic.

Synovial Membrane

It lines the inner surface of the fibrous capsule of the TMJ and the margins of articular disk. However, it does not cover the articular surface of the joint except for the posterior bilaminar zone. The synovial membrane folds to form the synovial villi which project into the spaces. The synovial membrane is made up of an intimal layer of synovial cells from one to four cells in depth that rest over a loosely organized subintimal connective tissue layer that has an extensive plexus of small vessels and numerous fibroblasts, mast cells, and macrophages. In contrast to other secretory cell layers, the synovial layer lacks a distinct basement membrane separating it from the underlying connective tissues. This enables a rapid diffusion of substances in and out of the joint cavities and back into the circulatory or lymphatic systems. The synovial fluid is considered to be a dialysate of plasma. The synovial membrane consists of two layers, a cellular intima resting on a vascular sub intima. The vascular subintima blends with the fibrous tissue of the capsule cells, which does not form a continuous layer but show gaps between the cells. The sub-intima is a loose connective tissue layer rich in blood capillaries, scattered fibroblasts, macrophages, mast cells, fat cells and some elastic fibers which prevent folding of the membrane.

Two types of synovial cells are recognized: The type A cells are responsible for the synthesis and transport of hyaluronate and are involved in active phagocytosis of any particulate debris transported to them by synovial fluid circulation. They have prominent golgi apparatus when viewed by electron microscopy. The type B cells are responsible for synthesis and transport of proteins into the synovial fluid. They have a prominent endoplasmic reticulum when viewed by electron microscopy. Functionally, the synovial membrane produces the synovial fluids which serve to (1) lubricate

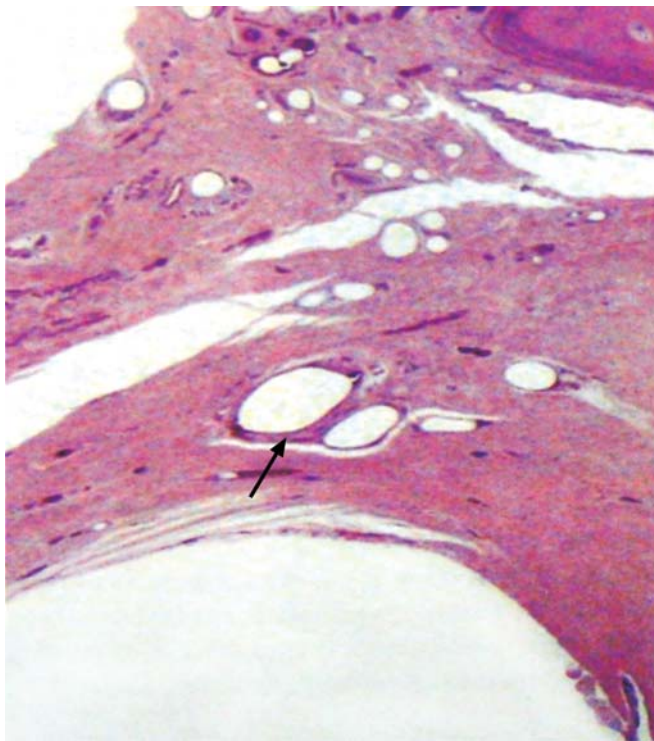


Fig. 17.10: Blood vessels (arrowed) in the posterior part of the articular disk

the joints surfaces during all joint movement; (2) provide essential nutrients for the chondrocytes within the cartilage matrix; (3) aid in the phagocytosis, and elimination of particulate and dissolved substances within the closed joint cavities; and (4) provide the necessary vehicle for transport and diffusion of substances in and out of the joint cavities and joint tissues. Lymphatic drainage occurs outside the synovial layer.

Synovial Fluid

Secreted by the synovial membrane, this straw colored synovial fluid is characterized by well-defined physical properties of viscosity, elasticity, and plasticity. It consists of a small population of varying cell types like monocytes, lymphocytes, free synovial cells and occasionally PMNs. The synovial fluid is a dialysate of plasma with some added proteins and mucin.

Functions of Synovial Fluid

- The synovial fluid acts as a medium for providing metabolic requirements to the avascular articular surfaces of the joint. Free and rapid exchanges exist between the vessels of the capsule, the synovial joint and the articular tissues.
- The serves as a lubricant and minimizes the friction between articular surfaces of the disk, condyle and fossa.

The synovial fluid lubricates the articular surface by way of two mechanisms, namely, boundary lubrication and weeping lubrication.

Boundary lubrication occurs when the joint is moved and the synovial fluid located in the border or recess areas is forced into the articular surface, thus providing lubrication.

- This type of lubrication prevents friction in the moving joint lubrication.
- This is the primary mechanism of joint lubrication.

Weeping lubrication refers to the ability of the articular surfaces to absorb a small amount of synovial fluid. Under compressive forces, a small amount of synovial fluid is released which acts as lubricant between the articular surface to prevent sticking.

- This helps to eliminate friction in the compressed but not moving joint.
- Only a small amount of friction is eliminated as a result of weeping lubrication.

ORIGIN AND EVOLUTION

The temporomandibular joint is a unique feature of the mammalian—no other vertebrates have it. Non-mammals use their mouth to capture food which they swallow whole. Mammals chew their food and require a different kind of TMJ.

The study of evolution of temporomandibular joint starts with the synapsids or mammal like reptiles. Some of the earliest reptiles, known to be present back in the Upper Carboniferous period (about 230 million years ago) belonged to the synapsids group. The synapsids continued upto the middle Jurassic period (about 160 million years ago). The synapsids survived for a period of about 200 million years from their first appearance until the end of lower Trias. They were the dominant group of animals at that period.

The early forms of synapsids are grouped as the Pelycosauria, which comprises carnivorous, piscivorous and herbivorous forms. The herbivores are of interest as they were the first group of vertebrates to exploit plant life directly as food. This is not possible in the sea where the plants are microscopic. But the first land vertebrate amphibia did not eat plant material as the amphibian did not have a fully developed dental apparatus.

In the Pelycosauria, the lower jaw is of an essentially reptilian form. The tooth-bearing bone or dentary has a strictly limited extension distal to the tooth row; the other bones of the jaw are well developed and comprise the posterior third of the jaw. The quadrate—the skull bone which forms, with the articular in the lower jaw, the jaw hinge—is a large bone with a considerable dorsoventral extension. The jaw joint is well below the level of the occipital condyle and there is no secondary palate, the internal nares being at the front of the buccal cavity. This implies that the food was not retained in the mouth to enable chewing to take place, as in mammals, but was swallowed fairly promptly after entering the buccal cavity. The herbivorous Pelycosauria would be, in fact, like the present day herbivorous lizards (e.g. Iguana).

In the carnivorous Pelycosauria, which include the ancestors of all the later mammal-like reptiles and also of the mammals, the teeth are all of a simple blade-like form. They have distal and mesial cutting edges, often serrated like a bread saw. In the carnivorous

Pelycosaurs, for example, the well-known *Dimetrodon*, the function of the dentition was to seize and kill the prey and then to cut off large lumps which would immediately be swallowed whole. Although the teeth are all similar in shape, they are different in size. Towards the anterior end of the maxilla, is a pair of teeth which are the largest in the jaw. These are replaced alternately, so that, while the norm is to have a pair of functional teeth in each maxilla during replacement, only one of the pair may be functional. These large teeth were concerned with the seizing and killing of the prey and may be called the canines. The teeth mesial to them on the maxilla and the premaxilla are the incisors. The teeth distal to them are the cheek teeth (we cannot use the term 'molars' and 'premolars' as these terms in mammals are defined in terms of tooth replacement). Thus, the functional differentiation of the teeth into incisors, canines and cheek-teeth had already taken place in the mammalian ancestors as early as the Lower Permian, although the differentiation of the teeth was really only a size difference, not a differentiation of morphological patterns.

More advanced mammal-like reptiles, which included both carnivorous and herbivorous forms replace the Pelycosaurs by the Upper Permian. There are two carnivorous groups in the Upper Permian, the *Terocephalia* and the *Gorgonopsia*. Both of these are more advanced than the Pelycosaurs in possessing, at least an incipient secondary palate and in showing a great reduction in the depth of that part of the skull which lies below the foramen magnum, so that the quadrate is greatly reduced in length. The next mammalian characteristic is that in placental mammals, all the teeth except the molars are replaced once, and only once. In these mammal like reptiles, the number of times the upper canine was replaced was similarly limited and fixed; the fact that replacement takes place once in mammals and more than once in the *Gorgonopsia* and *Terocephalia*, does not obscure the essential similarity of the process in both. Another change, also significant for the future, has taken place in the lower jaw. Although the other bones of the lower jaw are still large and well-developed the dentary has grown back over them dorsally to form a rudimentary coronoid process. To this attached the temporalis muscle would have been attached which has become large and important in these mammal-like reptiles, as is shown by the greatly

expanded temporal fossa. A powerful temporalis muscle is characteristic of mammals, particularly carnivorous ones.

In the Lower Trias period, the *Gorgonopsia* and the *Terocephalia* were replaced as the dominant carnivorous mammal-like reptiles by two other groups, the *Cynodontia* and the *Bauriomorpha*. The cynodonts and bauriomorphs of the Lower Trias are more mammal-like in several ways than are the gorgonopsids and therocephalians of the Upper Permian. In both, there is a secondary palate—better developed in the cynodonts, where it involves the palatine—and the postcanine teeth are no longer simple and blade-like. Although the jaw joint is still between the articular in the lower jaw and the quadrate in the skull, the reptilian jaw-bones have undergone considerable reduction in size and lie, at the posterior end, in a groove in the dentary.

During the Trias, the mammal-like reptiles declined and by the Upper Trias they had been replaced as the dominant group of land animals by the dinosaurs. However, by the Upper Triassic, one or more of the groups of mammal-like reptiles had crossed the boundary between reptile and mammal, so that we know two groups of mammals in the Upper Trias. One group, exemplified by *Kuehneotherium*, is the ancestor of almost all living animals; and the other, exemplified by *Morganucodon*, has as possible living descendants only the monotremes—the duck-billed platypus and the *Echidna* of the Australian region. The postcanine teeth in the *Morganucodon* differ from those in mammal-like reptiles and resemble the teeth of mammals in being elongated along the line of the jaw with two clearly separated roots. The teeth functioned as a shearing mechanism. Now, while the cheek teeth in mammal-like reptiles may have complex and broadened crowns, the teeth do not become elongated in the mesiodistal direction and remain single-rooted (the highly specialized, herbivorous tritylodonts are an exception which is not relevant here). The carnivorous mammal like reptiles did not in fact shear their prey as do modern carnivorous and insectivorous mammals or as did *Morganucodon* and *Kuehneotherium*. In such a shearing bite, the food being cut forms a wedge which tends to force the teeth apart. This is prevented by the action of the masticatory muscles holding the teeth in active occlusion. Thus, only the cheek teeth on one side of the jaw can be effective

at any one time, and each side has alternate periods of activity and rest.

The shearing bite also applies a twisting motion to the whole jaw, tending to dislocate the articulation. This is the reason for the glenoid in modern carnivores to extend laterally with a backwardly-directed process at its lateral end. The last is a thrust-bearing process, to resist the forces trying to dislocate the condyle by rotating it in the horizontal plane. The shrew has a double condyle on the dentary for similar reasons. Relative to the size of the animal the accessory jaw bones are as large in Morganucodon as in one of the later, carnivorous cynodonts, such as *Cynognathus*, and like the cynodont the mammal has a strong joint between articular and quadrate. This reptilian joint, however, was directly in the line of the tooth row and was not able to resist the twisting movement at the hinge produced by the shearing action of the teeth. To resist this, the second articulation was formed by a condyle on the end of the dentary working in the glenoid facet on the squamosal. The effect of this was to extend the articulation lateral to the tooth row, thus enabling it to resist the forces at the hinge tending to dislocate the jaw. The primitive reptilian jaw and quadrate articulation functioned as a simple lever and was weak. The primary reason for the evolution of the temporomandibular joint must have been to enable the jaw-articulation to resist the forces produced by the shearing dentition.

Once the temporomandibular joint had been established, it soon took over the whole of the jaw articulation, thereby releasing the quadrate-articular joint to pass into the middle ear. Changes in the muscle attachments and in the direction of pull have resulted in the reduction of the transmission of forces to the joint. Instead, it is delivered to the opposing tooth surfaces. The important reason for reduction in joint forces is the enlargement of coronoid process. The enlarged coronoid process along with the masseter relieves the temporomandibular joint of pressure and protects it from dislodgement and damage during eating.

CONDYLAR GROWTH AND GLENOID FOSSA DISPLACEMENT DURING GROWTH AND IN MALOCCLUSION

The condyle, a well established mandibular growth site, serves as the primary focus of functional orthopedic

therapy to stimulate or restrict mandibular growth. As the glenoid fossa determines the posterior/superior limit of the mandible, it also holds important implications for mandibular displacement. Bjork in 1963, observed condylar growth of 3 mm per year during the childhood period, a slight decrease to a prepubertal minimum, followed by an adolescent spurt peaking at 5.5 mm per year at approximately 14.5 years of age. Hägg and Attstrom, in their study observed greater condylar growth before (11.3 mm/3 years) than after the pubertal peak (9.6 mm/3 years). In normal growth, Bjork indicated that the distance between the fossa and nasion increases 7.5 mm between 12 and 20 years of age when the landmark articulare (AR) is used. Concurrent with the elongation of the posterior cranial base, the fossa and the temporal bone are displaced inferiorly and posteriorly. Such movements are important because the direction of fossa displacement in treated patients has been related to their overall growth patterns. Buschang and Ary concluded from their study after using condylion instead of articulare as reference point, the following:

- Posterior glenoid fossa displacement was almost twice as great as posterior condylar growth.
- Vertical condylar growth was approximately nine times greater than posterior condylar growth.
- Superior condylar growth and inferior fossa displacement were greater in adolescent boys than in girls.
- Articulare (AR) systematically overestimates inferior fossa displacements, underestimates superior condylar growth, and overestimates posterior condylar growth.

The varied position of glenoid fossa is linked to different malocclusions. Proper knowledge about normal condyle growth and fossa displacement will help in differentiating the growth changes from the changes produced due to growth. The condylar head inclination and superior joint space proved to be the most significantly correlated condylar characteristics to facial morphology. Patients with vertical facial morphology displayed decreased superior joint spaces and posteriorly angled condyles, whereas, patients with horizontal facial morphology demonstrated increased superior joint spaces and anteriorly angled condyles (Gail Burke et al, 1998).

The skeletal and TMJ structural changes resulting from chin cup application were investigated by analysis of cephalograms and cephalometric laminagraphs and

compared with those in control subjects by Hiroshi Mimura, and Toshio Deguchi. It was found that the chin cup changed the direction of growth of the mandible, and a definite ramus swing-back was seen.

The mandibular neck in the chin cup group was relatively more slender than that in the control group. The condylar heads were bent forward after the chin cup application, the glenoid fossa was deepened and widened, and the clearance between the condyle and fossa was decreased by the orthopedic chin cup force.

Harvold found that treatment with the types of activator used was found to effect a significant increase in mandibular alveolar height in the molar region and a reduced forward growth of the entire maxilla. This resulted in a transformation of the class II molar relationship into a class I molar relationship. The overjet was decreased by the reduced forward growth of the maxilla in combination with lingual tipping of the maxillary incisors. The growth in length of the mandible did not appear to be significantly influenced by the treatment.

In an experimental study of animals (McNamara), a proliferative chondrogenic response accompanied by deposition of new bony trabeculae at the bone-cartilage interface was evident. These morphologic changes in the condylar cartilage resemble in form, although not in magnitude, the changes previously documented in growing monkeys. In the growing monkeys, this chondrogenic response has been generally interpreted as an indication of the potential of the condylar cartilage to undergo compensatory growth as a means of re-establishing functional equilibrium.

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Myology and Neuromuscular Reflexes

CHAPTER OUTLINE

- **Muscles—Classification, Types**
- **Skeletal Muscle—Parts, Histology, Physiology of Muscle Contraction**
- **Development of Muscle and Adjustments During Growth**
- **Skeletal Muscles in the Craniofacial Region—Facial Muscles, Muscles of Mastication, Suprahyoid Muscles**
- **Form and Function**
 - Short face syndrome
 - Long face syndrome
- **Research Methodology regarding Form and Function**
 - Role of masticatory muscle thickness
 - Role of bite force
 - Role of spatial orientation of muscle
 - Role of muscle activity
 - Role of muscle fiber type
 - Buccinator mechanism
 - Muscle function in normal occlusion
- **Reflex Control of Jaw Muscles**

The internal structure of bone is constantly adapting to its functional environment through processes that remove existing bone and deposit new bone. The muscles provide an important mechanical stimulus for bone formation. A number of clinical and animal studies suggest a relationship between the masticatory muscle function and skeletal adaptation in the craniofacial region. A number of appliances used in orthodontics displace the mandible forward or downward, causing stretching of the orofacial soft tissues. An example is the bite block, which, when constantly present between the upper and lower dentitions, is used to disengage the occlusion or inhibit tooth eruption.

Optimal masticatory muscular force during growth is necessary for normal mandibular growth. Increase in muscle force will lead to a short face while decrease in

muscle force will lead to a long face. It is very important to understand the growth changes while the patient is growing such that interception of abnormal jaw growth is possible.

Various authors have reported that changes in dietary habits have led to changes in maxillofacial morphology and there is evidence of an evolutionary smaller jawbone in children. It is clinically important to understand changes in the facial growth due to dietary habits too.

MUSCLE

Muscle is a contractile tissue of the body and is derived from the mesodermal layer of embryonic germ cells. Muscle cells contain contractile filaments that move past each other and change the size of the cell.

The following functions can be attributed to muscles:

- Contraction for locomotion and skeletal movement.
- Contraction for propulsion.
- Contraction for pressure regulation.

Classification of Muscle

Muscle tissue may be classified according to a morphological classification or a functional classification.

- *Morphological classification (based on structure):* There are two types of muscles based on the morphological classification system:
 1. Striated
 2. Non-striated or smooth.
- *Functional classification:* There are two types of muscles based on a functional classification system:
 1. Voluntary
 2. Involuntary.

Types of Muscle

There are generally three types of muscles in the human body:

Skeletal muscle: This is striated and voluntary. Skeletal muscle or “voluntary muscle” is anchored by tendons to the bone and is used to affect skeletal movement such as locomotion and maintaining posture. Though this postural control is generally maintained as a subconscious reflex, the muscles responsible also react to conscious control like non-postural muscles.

Smooth muscle: This is striated and involuntary. Smooth muscles or “involuntary muscles” are found within the walls of organs and structures such as the esophagus, stomach, intestines, bronchi, uterus, urethra, bladder, blood vessels, and even the skin (in which it controls erection of body hair). Unlike skeletal muscles, smooth muscles are not under conscious control.

Cardiac muscle: This is non-striated and involuntary. Cardiac muscle is also an “involuntary muscle” but is more akin in structure to skeletal muscle, and is found only in the heart.

SKELETAL MUSCLE

Skeletal muscle is a type of striated muscle, usually attached to the skeleton. Skeletal muscles are used to create movement, by applying force to bones and joints; via contraction. They generally contract voluntarily (via somatic nerve stimulation), although they can also contract involuntarily through reflexes. The whole muscle is wrapped in a special type of connective tissue, epimysium. Skeletal muscles have one end (the “origin”) attached to a bone closer to the centre of the body's axis and this is often but not always a relatively stationary bone (such as the scapula) and the other end (the “insertion”) is attached across a joint to another bone further from the body's axis (such as the humerus). Contraction of the muscles causes the bones to rotate about the joint and to move the bones relative to one another.

Muscles have three major areas:

1. A belly or Gaster.
2. An origin: a tendinous connection of the muscle to a bone, usually the bone that is stabilized.
3. An insertion: a tendinous connection of the muscle to a bone, usually the bone to be moved.

Histology

Skeletal muscle is designed as a bundle within a bundle arrangement (Fig. 18.1). Individual muscle fibers are surrounded by endomysium. Muscle fibers are bound together by perimysium into bundles called fascicles; the bundles are then grouped together to form muscle, which is enclosed in a sheath of epimysium. At the ends of the muscle, all of the connective tissue sheaths (epimysium, perimysium, and endomysium) converge to form a tendon which will connect the muscle to its attachment site. Muscle spindles are distributed throughout the muscles and provide sensory feedback information to the central nervous system.

Types

Skeletal muscle fibers can generally be classified into two groups.

- Slow-twitch, or type I, fibers (sometimes referred to as “Red”) have more mitochondria, store oxygen in myoglobin, rely on aerobic metabolism, have a greater capillary to volume ratio and are associated with endurance; these produce ATP more slowly. Marathon runners tend to have more type I fibers, generally through a combination of genetics and training.
- Fast-twitch, or type II, fibers (sometimes referred to as “White”) have fewer mitochondria, are capable of more powerful (but shorter) contractions, metabolize ATP more quickly, have a lower capillary to volume ratio, and are more likely to accumulate lactic acid. Weightlifters and sprinters tend to have

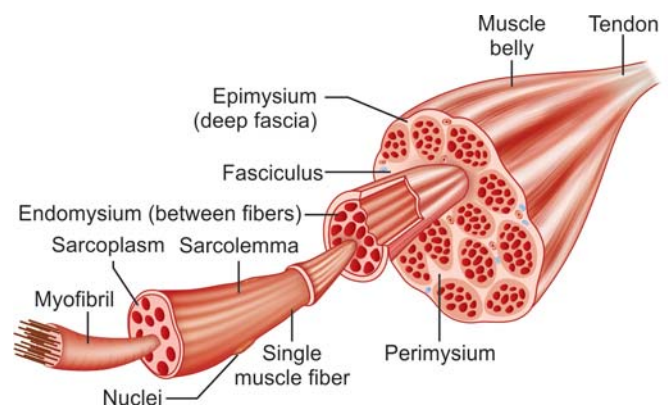


Fig. 18.1: Bundle within bundle arrangement: epimysium, perimysium, and endomysium are shown covering the muscle, fascicle and muscle fiber respectively

more type II fibers. Type II fibers are distinguished by their primary sub-types, IIa, IIx, and IIb.

The Muscle Fiber (Muscle Cells)

Skeletal muscle cells are elongated or tubular. The nuclei of these muscles are located in the peripheral aspect of the cell, just under the plasma membrane, which vacates the central part of the muscle fiber for myofibrils. Each muscle cell contains all the organelles that we find in other cell types. Although these organelles are the same as in other cells they are given special names.

The nucleus contains the genetic material of the muscle cell. The sarcolemma is the name given to the plasma membrane of the muscle cell. There are specialized invaginations of the sarcolemma that run transversely across the cell. These invaginations are known as T tubules (short for transverse tubules). The T tubules are essential for carrying the depolarization brought to the cell by a motor nerve impulse down into the muscle cell where it can have an effect on the terminal cisternae. The cytosol is the cytoplasm of the muscle cell and in the cytosol sarcoplasmic reticulum is found which is the endoplasmic reticulum of the muscle cell. There are sac-like regions of the sarcoplasmic reticulum known as terminal cisternae. The terminal cisternae act as calcium storage sites. The calcium ions stored in the terminal cisternae are essential in muscle contraction. In skeletal muscles two terminal cisternae are associated with a T tubule to form a structure known as a triad. Mitochondria are sites of energy production (ATP synthesis) in the muscle cell as in all other cells of the body.

A myofibril is a cylindrical bundle of contractile proteins found within the muscle cell. Note that there are several myofibrils within each muscle cell. It is the arrangement of the contractile proteins within the myofibril that causes the striated appearance of skeletal and cardiac muscle. Myofibrils are composed of individual contractile proteins called myofilaments. These myofilaments are generally divided into thick and thin myofilaments. The thin myofilaments are composed mainly of a protein known as actin. Actin filaments are anchored into the Z-line of a sarcomere. The thick myofilaments are composed mainly of the protein myosin. It is the orderly overlapping of the actin and myosin filaments that give skeletal muscle their striated

appearance (light and dark bands) (Fig. 18.2). The 'A' band is the dark band and corresponds to the length of a bundle of myosin filaments. As muscle contraction is a sliding of the myofilaments past each other, we do not see any of the myofilaments actually shorten. However, the width of the banding patterns change as the degree of overlap changes. Since the 'A' band corresponds to the length of the myosin filaments, and these filaments do not shorten, the width of the 'A' band also does not shorten.

The light bands are known as I bands. The I bands are composed mainly of actin filaments. Each I band is bisected by a protein disk known as the Z-line. Actin filaments are anchored into the Z-line. During muscle contraction, the actin filaments slide over the myosin filaments which results in a shortening of the I band.

In the middle of the 'A' band is a somewhat lighter area known as the H zone. This zone corresponds to the area where we have myosin not overlapped by actin (the area between the thin filaments). During muscle contraction, the actin sliding over the myosin encroaches into this area so that the H zone shortens. In the middle of the H zone, we see a dark band known as the M line. The M line is comprised of protein fibers that function to anchor the myosin filaments.

The area between two Z lines is known as a sarcomere. The sarcomere is the functional or contractile

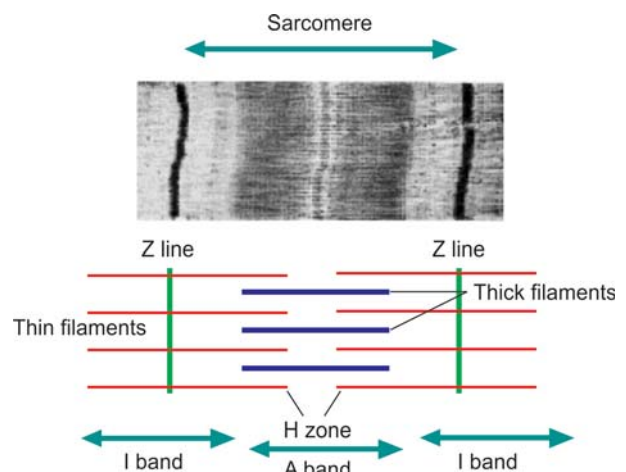


Fig. 18.2: The unit between two consecutive Z-lines is defined as the sarcomere and it is the basic contractile unit of the myofibril. The thick filaments of myosin form the A band which is visible as the striations. The thin filaments are predominantly composed of actin polymers

unit of muscle. To summarize, a whole muscle is made up of many smaller bundles known as fascicles. Each fascicle is made up of many muscle cells (myofibers). Myofibers contain cylindrical bundles of myofibrils which in turn, contain many smaller bundles of myofilaments (Fig. 18.3).

Physiology of Muscle Contraction

Muscles contract when they receive a motor impulse from a motor nerve. These nerve impulses serve only a limited number of muscle fibers. The muscle fibers served by a single motor neuron make up a structure known as a motor unit. Motor units allow for selective contraction of muscle fibers so that we may control the strength and extent of muscle contraction. Without motor units, a nerve impulse to the muscle would result in the entire muscle contracting to its full extent. That would make every motion that we make an "all or none" motion. This type of movement would make life nearly impossible.

The main theory of muscle contraction used today is based upon Huxley's *sliding filament theory*. The key principle behind this process is the overlapping of the actin and myosin filaments. This leads to the shortening or closing up of the sarcomere thus leading to a muscular contraction. The thin actin filaments are surrounded by two substances, troponin and tropomyosin. Troponin is a globular protein complex that at rest holds the tropomyosin in place and blocks the myosin binding sites

upon the actin filaments. Tropomyosin is more of a thin wire like strand which has troponin attached to it at regular intervals.

When the body requires a movement, an electrical signal will be sent from the brain or spinal cord. This signal releases the neurotransmitter acetylcholine (ACh). This is sent down to the muscle and stimulates the release of calcium. There will be a sudden release of calcium from special 't' vessels which are inside the sarcoplasmic reticulum. Troponin has a great affinity (likening/attraction) for the calcium. When the calcium makes contact and binds with the troponin, there will be a change in shape of both the troponin which tropomyosin. The calcium binds to the troponin and leads to a movement. As the troponin is moved, it brings the tropomyosin with it, thus exposing the binding sites upon the actin. At the same time, the heads of the myosin filaments will become activated by the ATP (adenosine triphosphate).

The ATP is then broken down to form ADP (adenosine diphosphate), one free phosphate molecule and releases a large amount of energy thus allowing the myosin heads to move. The myosin heads now attach themselves to selected sites or places upon the actin filaments to form actin-myosin bonds. This is usually referred to as the cross bridge.

This cross bridge operates in different phases so that at any given time, only half of the myosin heads will be attached to the actin. This allows a constant pulling

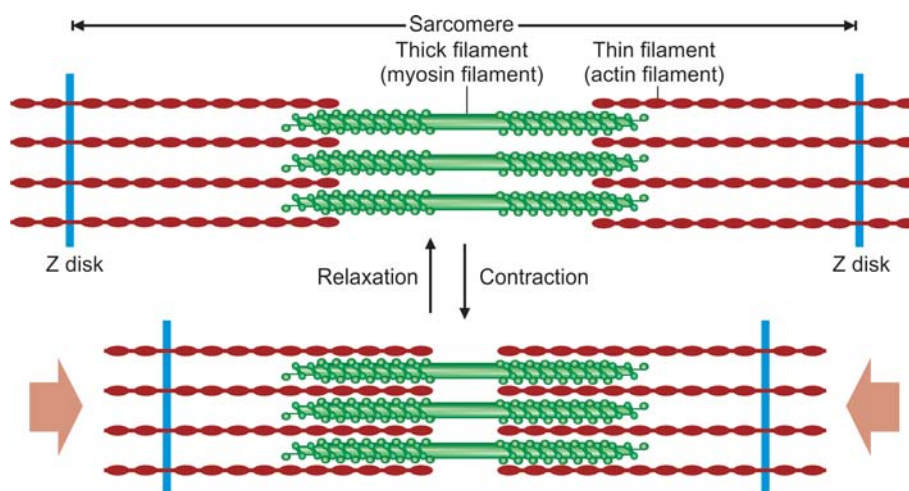


Fig. 18.3: Illustration of dimensions of sarcomere during muscle contraction. The Z lines move closer to each other. Note that the light I bands and the H zone become wider, but the width of the dark A band remains unchanged

of the actin and myosin closer together. This cross bridge is immediately followed by a detachment and reattachment to a point further along the actin filament and so on. The movement of attachment, detachment and reattachment is known as the *ratchet mechanism* due to its constant pulling of the fibers. So while the contraction continues, an enzyme known as myosin-ATPase is released upon the cross bridge being formed which catalyses the release of energy. This release of energy is actively used to allow the constant pulling of the fibers in one direction. This results in a shortening of the sarcomere. The overall goal of this is to pull the actin filaments past the myosin filaments forming a greater overlap between the actin and myosin (greater than at resting). This mechanism is carried out in thousands of sarcomeres along each muscle fibre and the overall shortening is required in a number of muscle fibers to give a contraction, thus leading to a bodily movement. This contraction continues while there is sufficient calcium available.

An isometric contraction (no change in muscle size) is slightly different from this. The muscle fibers are held at a certain point of stretching with the actin and myosin bond occurring, thus leading to an isometric contraction.

DEVELOPMENT OF MUSCLE AND MUSCLE CHANGES DURING GROWTH

Prenatal muscles grow by increase in size and amount of fibrous tissue surrounding the muscle bundles, as well as by cell division. Striped muscle differentiation begins in the 7th week of intrauterine life and typical muscle fibers are seen in the 22nd week. Normal muscular activity begins at the end of the 7th month and is not complete in the extremities until after birth. Muscles of mastication at first develop in relation to Meckel's cartilage but are independent of the insertions and are attached only to the forming mandible. Increase in bulk of a muscle is due to activity. Atrophy results from disuse. During infancy and childhood, gain in muscle tissue is essentially the result of hypertrophy. Between the 4th fetal month and birth, the muscular system increases by 50 fold. It increases 40 fold between birth and middle of the 3rd decade of postnatal life.

Postnatal growth: Muscle growth is rapid in infancy and childhood, slower and regular in the middle of childhood and again more rapid preceding and during adolescence.

The muscles of the head show the smallest relative increment of growth. The weight of the facial musculature increases 4-fold between birth and age 20 years, while that of the mandible alone increases almost 7-fold by age 20 years.

Adjustments during growth: Continued adjustments in muscle attachments occur during skeletal growth. Muscles can be divided into two groups with respect to their attachments.

Periosteal: Muscles attached to the fibrous layer of the periosteum.

Tendinous: Muscles attached by means of tendon which cannot be removed from the bones without some destruction of the surface of the bone.

The first group can shift its attachments by growth changes of the periosteum. Different rates of lengthening at different regions allow the periosteum to shift relative to the bone, carrying the muscle attachments with it thus maintaining the constant spatial relationship of the muscles.

In the second type of muscle attachments a mechanism exists to break down or alter the attachment so that the muscles may shift. In muscles attached by tendons, the change is made by bone resorption and apposition, which carries the tendinous attachments with it. The insertion of the suprahyoid and external pterygoid muscles into the mandible belong to the second group and to a certain extent also the internal pterygoid and temporal muscles since their insertions are partly tendinous.

Where bone resorption is found in relation to the tendinous attachment of a muscle, resorption frees the muscle from the bone. Muscles can become temporarily periosteal in attachment and can shift relative to bone growth, maintaining their normal position. This is particularly true of muscles attached at the growing ends of the mandible. When bone resorption ceases, the muscles may become reattached directly to the bone by tendinous fibers.

Growth at the anterior end of each half of the mandible until the symphyseal suture is obliterated in the latter part of the first year, and gradually tends to separate the anterior belly of the digastric and the geniohyoid muscles. The tendinous insertion of the temporal muscle is gradually fixed from the bone of the

anterior border of the ramus of the mandible which is resorbed to make room for eruption of permanent molar and the development of the alveolar process around these teeth. The attachment of internal pterygoid shifts during the growth of the mandible and expands as the ramus increases in size by bone deposition along its posterior border.

SKELETAL MUSCLES IN THE CRANIOFACIAL REGION

Facial Muscles (Fig. 18.4)

The primary function of facial muscles is the expression of emotions. The capacity for expressing effective states is highest developed in the human. Coleman contends that the average human is capable of 7000 possible facial expressions. In addition to expression of emotions, these muscles are important in the maintenance of posture of facial structures. According to Proffit, the lip and buccinator muscles opposed by the tongue contribute to a postural equilibrium of the teeth. The facial muscles also contribute to stabilization of the mandible during infantile swallowing and in chewing and swallowing in the edentulous and occlusally compromised adult. It is quite possible that postural alternations in the facial muscles may contribute to structural changes in the jaws. Frankel has speculated that the buccinator muscles exert

a constraining force on the maxillary alveolar process as well as the teeth. Form also dictates function: patients with short upper lips or excessively proclined maxillary incisors compensate by the elevation of the lower lip through the action of the mentalis muscle to establish an anterior seal during swallowing. Facial muscles also play an important role in both visual and spoken communication. Lips and cheeks are essential for bolus control in mastication as well. The effects of actions of various facial muscles are given in Table 18.1.

Muscles of Mastication (Figs 18.5A and B)

The chief muscles of mastication are: masseter, lateral pterygoid, temporalis and medial pterygoid.

The Temporalis Muscle

This is an extensive fan-shaped muscle that covers the temporal region. It is a powerful masticatory muscle that can easily be seen and felt during closure of the mandible.

Origin: floor of temporal fossa and deep surface of temporal fascia.

Insertion: tip and medial surface of coronoid process and anterior border of ramus of mandible.

Innervation: deep temporal branches of mandibular nerve (CN V³).

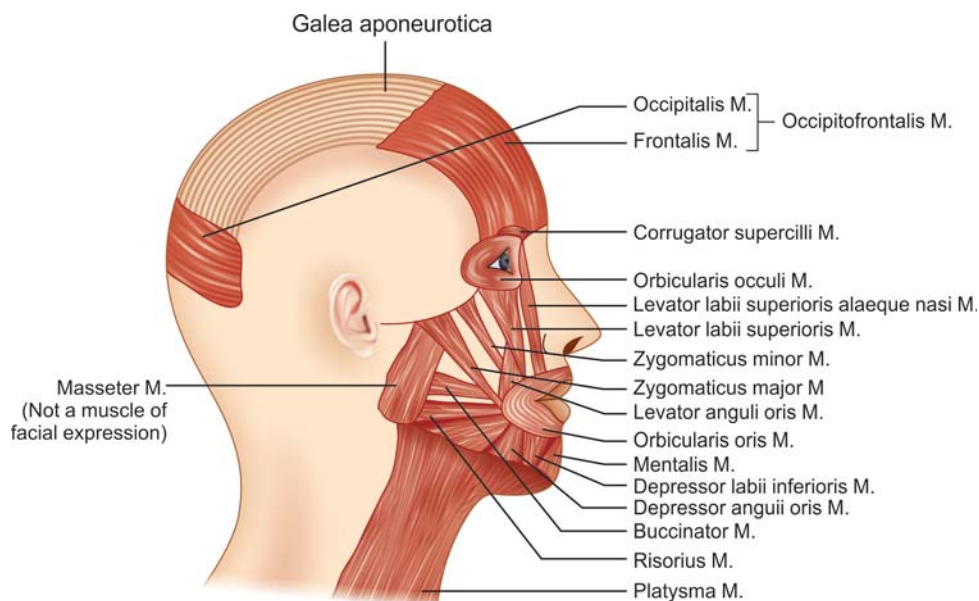


Fig. 18.4: Facial muscles

Table 18.1: Facial muscles and their effects on facial expression

Action	Muscle name	Effect
Raise inside brow L/R	Frontalis medial portion	Many expressions
Raise outside brow L/R	Frontalis lateral portion	Many expressions
Tighten inside brow frown	Corrugator supercilli + Procerus	Anger, pain, disgust
Eyes wide L/R	Levator palpebrae superioris	Surprise, fear, shock
Eye squint L/R	Orbicularis oculi orbital portion	Anger, thought, concentration
Eyelid close L/R	Orbicularis oculi palpebral portion	Blink, wink
Nostril flare L/R	Dilator Naris + Levator Labii superioris alaeque nasi	Disgust
Purse lips	Incisivus labii	Kiss, anger, "oo", whistle
Smile corner L/R	Zygomaticus major	Smile
Corner mouth down into sadness L/R	Depressor anguli oris + Zygomaticus minor + Depressor anguli oris + Mentalis	Sadness
Top lip up L/R	Levator labii superioris	Disgust, part lips for sounds
Lower lip down L/R	Depressor labii inferioris	Part lips for sounds
Tighten lips U/L	Orbicularis oris	"p", "b", "m" anger
Jaw open	Digastric	Speaking, surprise
Jaw slide L/R	Masseter	Slide jaw L/R

Action: The temporalis muscle elevates the mandible, closes the jaws; and its posterior fibres retrude the mandible after protrusion.

The Masseter Muscle

This is a quadrangular muscle that covers the lateral aspect of the ramus and the coronoid process of the mandible.

Origin: inferior border and medial surface of zygomatic arch.

Insertion: lateral surface of ramus of mandible and its coronoid process.

Innervation: mandibular nerve via masseteric nerve that enters its deep surface.

Action: It elevates and protrudes the mandible, closes the jaws and the deep fibres retrude it.

The Lateral Pterygoid Muscle

This is a short, thick muscle that has two heads or origin. It is a conical muscle with its apex pointing posteriorly.

Origin: superior head—infratemporal surface and infratemporal crest of the greater wing of the sphenoid bone, inferior head—lateral surface of lateral pterygoid plate.

Insertion: neck of mandible, articular disc, and capsule of temporomandibular joint.

Innervation: mandibular nerve via lateral pterygoid nerve from anterior trunk, which enters it deep surface.

Acting together, these muscles protrude the mandible and depress the chin.

Acting alone and alternately, they produce side-to-side movements of the mandible.

The Medial Pterygoid Muscle

This is a thick, quadrilateral muscle that also has two heads or origin. It embraces the inferior head of the lateral pterygoid muscle. It is located deep to the ramus of the mandible.

Origin: deep head—medial surface of lateral pterygoid plate and pyramidal process of palatine bone, superficial head—tuberosity of maxilla.

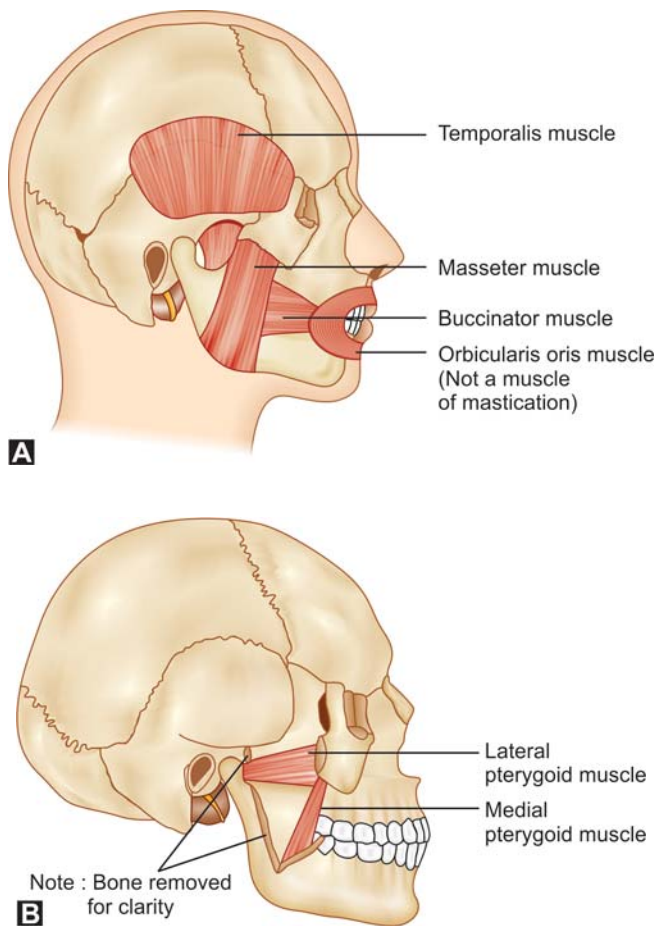
Insertion: medial surface of ramus of mandible, inferior to mandibular foramen.

Innervation: mandibular nerve via medial pterygoid nerve.

It helps to elevate the mandible and closes the jaws. Acting together, they help to protrude the mandible. Acting alone, it protrudes the side of the jaw. Acting alternately, they produce a grinding motion.

FORM AND FUNCTION

Embryologically, the bones that make up the maxillofacial region are membranous bones and are more susceptible to the environmental factors such as the stimulating influence of muscles and the extrafunctional force in comparison with the long bones of extremities which are formed by cartilaginous ossification. Skeletal growth to a considerable extent is influenced by muscular growth, particularly, the parts of bones to which muscles attach, develop in conjunction with the muscle (coronoid process). The muscles might not act by inducing growth



Figs 18.5A and B: Muscles of mastication

at the area of muscle attachment. For example, the gonial angle area of the mandible is strongly influenced by growth of the elevator muscles of the mandible. The mandibular ramus does not grow by lengthening of the ramus from apposition of bone at the gonial angle, which might be expected if lengthening of the muscle directly produced new bone in that area. Instead, the gonial angle area is often resorptive, while mandibular length is produced through proliferation at the condyle and along the posterior border of the ramus in areas away from the muscle attachment.

The first evidence of muscles influencing skeletal growth was the discovery of the trabecular alignment inside the femur head. The femur head had the stress trajectories formed in resistance to functional stresses. The stimulating influence of muscle or extra-functional force produced demonstrable changes in bone. Thus the shape and internal structure of the femur head are

related to lower extremity function. This theory is recognized as Wolff's law. Later on functional matrix theory of Melvin Moss explained the mechanism by which the soft tissue envelope could direct/divert the skeletal growth. Sassouni (1969) outlined the concept that the vertical alignment (and subsequent force) of jaw-closing muscles direct skeletal growth toward a shallow mandibular plane angle, an acute gonial angle, and deep bite, whereas obliquely aligned jaw-closing muscles (with subsequent diminished force) permit a steep mandibular plane, an obtuse gonial angle, and open bite. He classified the skeletal facial types into short face syndrome and long face syndrome.

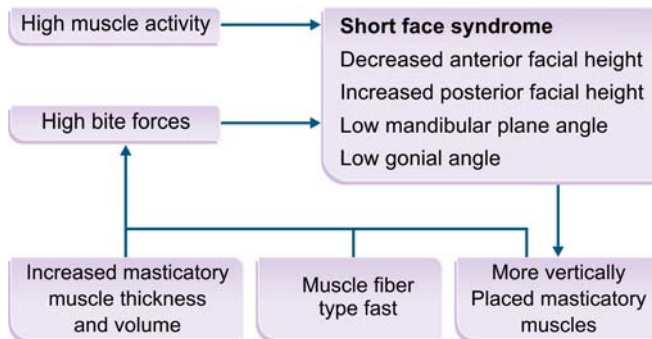
Short Face Syndrome (Fig. 18.6)

The short face syndrome (SFS) is a clinically recognizable facial type with reduced lower facial height as the common denominator. The characteristics include reduced eruption of posterior teeth, increased posterior facial height, and a flat mandibular plane angle. Two subgroups were distinguished in the short face syndrome group. SFS1 is characterized by a long ramus, sharply reduced SN:MP angle, and a slightly reduced posterior maxillary height. In contrast, SFS2 is characterized by a short ramus, a slightly reduced SN:MP angle, and a sharply reduced posterior maxillary height. The latter group was designated as vertical maxillary deficiency. The various factors contributing to horizontal growth is given in the Flow chart 18.1.

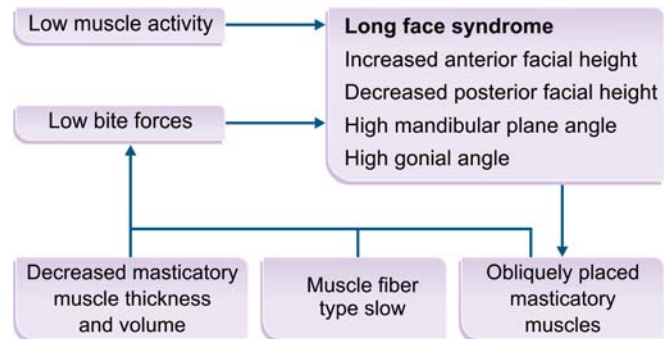


Fig. 18.6: Cephalogram of a patient with short face syndrome

Flow chart 18.1: Factors leading to increased horizontal growth of the facial skeleton



Flow chart 18.2: Factors leading to increased vertical growth of facial skeleton



Long Face Syndrome (Fig. 18.7)

An important aspect of comprehensive orthodontic therapy is managing the vertical dimension of the patient's face. Many of the most difficult orthodontic cases involve long face syndrome. Characteristic features include excessive eruption of posterior teeth, normal or excessive eruption of anterior teeth, short posterior facial height, and a steep mandibular plane angle. Common diagnostic criteria for long face syndrome include the gonion (Go) to gnathion (Gn) to sella-nasion (SN) line angle (mandibular plane angle) of 37° or greater, and a posterior (S to Go) to anterior facial height (N to menton (Me)) ratio of 0.65 or less. The primary cause of long face syndrome is an unfavorable growth pattern.



Fig. 18.7: Cephalogram of a patient with long face syndrome

Limiting the extrusion of the posterior teeth is critical in orthodontic management of long face syndrome. If the posterior teeth extrude more than normal, the bite opens. This increases the mandibular plane angle and reduces the ratio of posterior to anterior facial height. These changes cause characteristics of long face syndrome. As the mandible opens along an arc, excessive bite opening results in a retruded mandibular position. Sassouni and Prah Anderson et al, showed that a retruded mandible combined with characteristics of long face syndrome results in poor facial esthetics. McNamara showed that more than 60 percent of patients with class II malocclusions exhibit one or more symptoms of long face syndrome. Factors responsible for vertical growth are enumerated in Flow chart 18.2.

RESEARCH METHODOLOGY REGARDING FORM AND FUNCTION—FACTORS CONTROLLING FACIAL GROWTH

Role of Masticatory Muscle Thickness

The thickness of the muscle is a variable that can be easily measured by a variety of techniques, since majority of muscles in the area of interest in the craniofacial region are superficial. Thickness of the masticatory muscles (especially masseter) have been measured and correlated with variables of facial morphology. Computed tomography, magnetic resonance imaging and ultrasonography (Fig. 18.8) are some of the commonly employed techniques. The masticatory muscle thickness increases with age. Males have thicker masticatory muscles when compared to females. The thicker the muscles, the more tension generated by them. The high maximal



Fig. 18.8: Thickness of the superficial muscles can be measured rapidly by diagnostic ultrasound. The thickness of masseter muscle—distance between the superficial fascia of masseter muscle and the lateral surface of the ramus of the mandible is measured in the above picture

bite forces, so derived are thought to control the facial growth. The masseter muscle in a long face is thinner by 30 percent. Thicker masseter muscle is found to significantly correlate with reduced gonial and mandibular plane angles and increased ramus height implying its role in the more horizontal development of face. The thicker masseter also leads to a broader maxillary arch and a broader face in general. Based on these findings, Ingervall proposed training the jaw muscles of long-faced children by having them chew daily on tough material to strengthen the muscles and to induce a more favorable anterior mandibular growth rotation. All this goes against the philosophy that the long-face pattern elevator muscles fail to gain strength in the mandible. It is the muscle which controls morphology not vice versa.

Role of Bite Force

When the morphology of the skulls from 17th and 18th century on hard diet were compared to the facial skeleton of living individuals, it was found that the skulls had less anterior facial height. Thus, it was postulated that subjects with a higher bite force have a relatively short lower anterior facial height. It was just beginning for a wide variety of bite force studies. Studies on humans typically use a bite fork which has a force transducer which is placed between the teeth and used to record the bite force. The cephalometric X-rays are used to record the

facial skeleton. Laboratory studies mainly use rats which are fed diets of varying consistencies. They are later sacrificed to facilitate direct measurement of their skeleton.

Masseter muscle is the most important contributor to the bite force. High bite forces are related to decrease in anterior facial height, gonial angle and mandibular plane angle. Bite forces alter the region which affects the occlusal loading thus inducing a change in the direction of growth.

Bite force magnitude is related to jaw muscle thickness, fiber type composition, sarcomere length, jaw muscle activation level, direction of bite, age, sex and occlusal contact measures. Other factors which seem to influence the bite force are the state of dentition, location within dental arch where force is measured, psychological and mental conditions during the effort, attitude of the investigator and the subject, malocclusions, presence of tenderness in the muscle and the extend of vertical separation of the teeth due to the bite fork. All these factors explain the broad range of variability of the results obtained in different bite force studies.

Role of Spatial Orientation of Muscles

Differences in facial morphology result in significant differences in the spatial orientation of the muscles which in turn determine the moment arm of the masticatory muscles. The dento-skeletal morphology has been shown to be related to masticatory muscle orientation in children. Short face types have more vertically placed masticatory muscles whereas long face types have more horizontally placed muscles. Therefore, there is a variation in the direction of bite force between long face and normal adults.

Van Spronsen et al (1997) studied the relationship between the orientation and moment arms of the human jaw muscles and normal craniofacial morphology. The anterior face height factor significantly correlated with the orientation of the jaw opening muscles in the sagittal plane but did not significantly correlate with the orientation of the jaw closing muscles. The sagittal moment arms of the mandibular elevators showed significant correlations with the factors describing the gonial angle and the posterior face height. It was concluded that the variation of spatial orientation of the human jaw closing muscles is predominantly associated with variation of mandibular morphology (expressed by

the gonial angle) and the posterior face height. The hypothesis that persons with an increased anterior face height have relatively oblique orientated jaw elevators was rejected. Subsequent research showed that the variation of the spatial orientation of the jaw muscles is small and does not significantly contribute to the explanation of the different molar bite-force levels of long face and normal subjects

Role of Muscle Activity

The role of muscle activity in controlling the craniofacial growth has been studied in normal subjects with surface electromyography. The instrument used for evaluating the activity of the orofacial muscle is the electromyograph. It is used to measure the electrical activity. Two types of electrodes are used. They are: surface electrode and needle electrode. Surface electrodes record from a larger population of muscle fibers than do needle electrodes. Both types of the electrodes record the membrane action potentials from several fibers in a single motor unit, which arrive at the electrode at different times giving a unique signature to that unit as long as the electrode is not moved. This permits the study to the behavior of individual units and how the units are recruited. A flat metal plate is placed over the muscle to be tested. Then, a thin sterile needle attached to wires of a recording machine is inserted through the skin into the muscle. The electrical activity of the muscle is recorded at rest and during contraction. It is then displayed as electrical waves on an oscilloscope and amplified to produce sounds over an audio speaker. The action potentials from the various units merge together and produce the typical electromyogram. Although EMG can give useful information on whether a muscle is active and defines when the activity begins and in the muscle fibers sampled, it is impossible to know how much of the muscle activity is being missed.

Surface electromyography (EMG) detects the firing of motor units which can be used to monitor muscle activity. EMG measurements have been taken at postural rest, chewing, swallowing and maximal bite (Fig. 18.9). Electromyographic studies, showed decreased activity in all jaw muscles in long-faced persons. Masseter and digastric activities are shown to have significant negative correlation with vertical craniofacial morphology. Mouth breathing is found to be associated with reduced EMG activity of masseter and could be responsible for the long

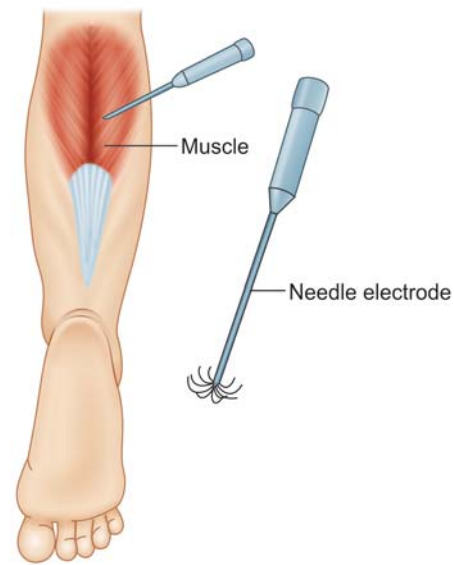


Fig. 18.9: Electromyography is the measurement of electrical activity that occurs within muscle fibers in response to nervous system stimulation. As muscles contract, electrical signals with amplitudes in the microvolts (millionths of a volt) range, are created within the muscles. Sensors placed on the skin's surface detect these electrical signals from the active muscles and provide this information to the EMG unit

face seen in such patients. High correlation between bite force and EMG activity of masseter is also observed. Short face types have high bite force levels and increase the EMG activity of masseter. Decreased jaw muscle activity has been demonstrated in long face subjects. Animal studies have supported EMG studies.

Many studies reported to date on facial morphology and EMG suffers from methodological limitations. Some, for example, use only a single measure of biting force, typically a maximum bite force, and these are compromised by unknown levels of subject motivation and short-term fatigue and pain. Others measure EMG activity during activities such as chewing, clenching, or swallowing that may differ considerably among subjects. Furthermore, few have controls for age or gender. Long time EMG activity registration is used in some studies.

Craniofacial morphology is determined by the activity of the jaw muscles and the muscle activity is affected in diseases like muscle dystrophies. Vertically oriented craniofacial growth has been described as a result of progressive atrophy of the jaw muscles. Masseter is affected by muscular dystrophy and the etiology of associated long face pattern is attributed to it.

Role of Muscle Fiber Type

It has been shown that type I fibers with slow shortening velocities produce less force per unit area than do the type II fibers with rapid shortening velocities. Hence, muscles with a high percentage of type I fibers are less powerful than muscles with predominantly type II fibers. Studies have found a significant positive correlation between molar bite-force and the proportion of type II masseter fibers. However, the studies have been sparse in this direction due to the following reasons:

- No consensus exists about the distribution and size of muscle fiber types in the jaw muscles of long-face subjects.
- Association between types of fibers in a given area of muscle cross section and the maximal tension developed by that unit was shown to be poor.
- It is proven that changes in the masseter fiber type seen in long face are due to primary myopathy than a reflection of functional requirements.

The facial growth patterns of a child are determined by the balance of different muscular influences around the surrounding cranial bones as well as predetermined genetic factors. The growth of face is affected by the varying grades of muscular activity round the clock including the heavy chewing forces exercised occasionally. The thick skeletal muscles can generate more muscular tension leading to a more horizontal rotation of mandible in turn leading to short face. Short face positions major jaw closing muscles vertically imparting to them a mechanical advantage which would further enhance growth in the same direction. The growth in the horizontal direction expands the palate broadening the maxillary arch. The opposite applies to the vertical growth pattern.

The muscles in the facial region contract most while chewing food. The consistency of the food also regulates the facial growth changes in a child as more force is required to chew raw food than refined food. The increased effort also causes the muscle to hypertrophy. The shift in food habits to more refined food stuffs is hypothesized to produce growth in more vertical direction.

Buccinator Mechanism (Fig. 18.10)

Muscles are a potential force whether they are at rest or in active function.

Teeth and supporting structure of the jaw are under the control of the adjacent muscles. The balance between the muscles is responsible for the integrity of the dental

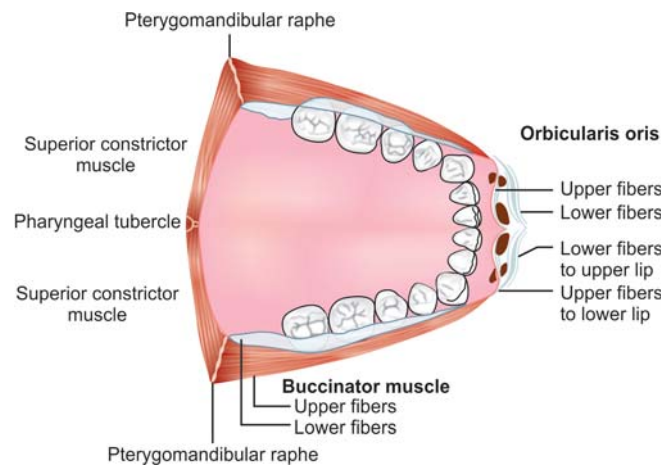


Fig. 18.10: Buccinator mechanism. Note the intermingling of fibers which act as one single powerful unit exerting contractile force on the arch

arches and the relation of teeth to the arches. Buccinator mechanism refers to a phenomenon in which a continuous band of muscles that encircle the dentition and is firmly anchored at the pharyngeal tubercle of the occipital bone. Buccinator mechanism starts with the decussating fibers of the orbicularis oris joining the right and left fibers of the lip which constitute the anterior component of the buccinator mechanism. It then runs laterally and posteriorly around the corner of the mouth, joining other fibers of the buccinator muscle which gets inserted into the pterygomandibular raphe. Here, it mingles with the fibers of superior constrictor muscle and runs posteriorly and medially to get fixed to the pharyngeal tubercle. All of these muscles, numbering thirteen with elasticity and contractility acts like a rubber band tightly encircling the bone system, the mandible. The tongue acts opposite to the buccinator mechanism exerting an outward force. The clinical significance of buccinator mechanism is that any imbalance in buccinator mechanism leads to malocclusion. In pernicious oral habits like thumb sucking and tongue thrusting, the equilibrium between buccinator mechanism and tongue is lost. This causes various changes in dentition like constricted maxillary arch, increased proclination and open bite.

Muscle Function in Normal Occlusion

Muscle function is usually normal in cases of class I malocclusion. The teeth are in a state of balance with environmental forces (Fig. 18.11). While the actual

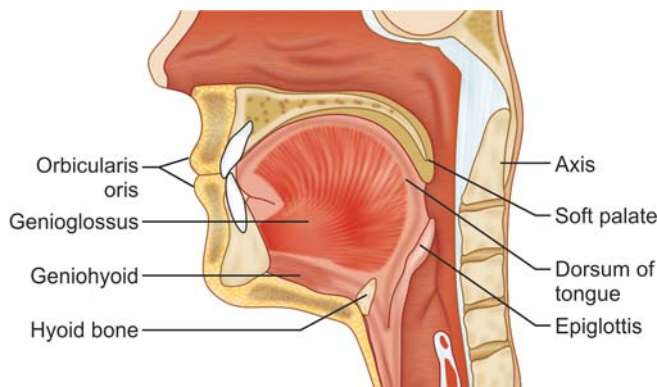


Fig. 18.11: Normal structural muscle relationship. Note proximity of tongue and palate; gentle, unstrained lip contact; normal overbite and overjet

measurements of tongue and lip forces show that they are not equal in any one area during a particular function, a state of equilibrium has been reached if we consider morphogenetic pattern, tooth size, available basal bone, and character of contiguous tissue, postural forces, and the various functional forces. Mastication is the primary consideration of dentists, when we think of teeth, jaws, and the motivating musculature. This is only part of the picture. Posture, deglutition, respiration, and speech make use of the same structures, and these functions are no less important. The head is balanced precariously on a bony column and is held erect by the chain of postvertebral, prevertebral, masticatory, facial, suprahyoid, and infrahyoid muscles.

REFLEX CONTROL OF JAW MUSCLES

Reflex is defined as *an automatic and often inborn response to a stimulus that involves a nerve impulse passing inward from a receptor to a nerve center and thence outward to an effector without reaching the level of consciousness*. A “reflex” in biology is generally taken to mean a behavior which does not require voluntary initiation, but occurs automatically under appropriate conditions. Even a unicellular organism will approach a light source when it is turned on, or avoid certain chemicals in its environment. While these acts may have adaptive value, and be retained by evolution, they are certainly far from conscious; they are more like what we mean by a “reflex”. Reflexes generally involve feedback of a signal, to maintain some condition in the body relatively constant. This is the basis of animal

homeostasis. It has a definitive neuronal connection known as reflex arc. A *reflex arc* is a chain of neural connections between the receptor and the effector. Its components are: sensory neuron, interneuron(s)—may have more than one or none, and motor neuron.

There are a number of ways of classifying reflexes. Reflexes can be classified in terms of the number of neurons or synapses between the primary afferent neuron and the motor neuron. We distinguish two types, the monosynaptic reflex and the much more common, multisynaptic or polysynaptic reflex. The term multisynaptic implies that more than one synapse is involved, whereas polysynaptic usually implies that the pathway is of variable length, some parts disynaptic, some trisynaptic, etc. Neural reflexes may be roughly categorized as (1) Postural, or antigravity; (2) Protective, i.e. withdrawal or avoidance; (3) Cardiovascular, for instance in regulation of arterial pressure; (4) Respiratory, either for airway protection or regulation of blood gas levels; (5) Digestive, either peristaltic (mechanical) or secretory (neurochemical); (6) Specialized, for functions such as pupillary constriction in the eye or sexual behaviors; and (7) Humoral, for instance in the thyroid and pituitary-adrenal systems.

The main reflexes associated with the jaw muscles are the postural or antigravity and protective, i.e. withdrawal or avoidance types. They share some characteristics with spinal reflexes affecting the arms and legs.

Receptors

- **Muscle spindles:** In humans, the jaw-closing muscles, not the jaw-opening muscles, contain muscle spindles. Muscle spindles are miniature muscles situated in the muscle belly of cross-striated skeletal muscles. Within the muscle spindle are a number of intra-fusal fibers that consist of contractile elements and are surrounded by a fusiform capsule. The muscle spindle is innervated by different sensory and motor neurons. The muscle spindle afferents start working when the middle part of the intra-fusal fibers is stretched. This can happen either by a passive stretch or by active shortening of the polar parts of the intra-fusal fibers. The latter takes place when the motor nerve fibers are stimulated. The motor nerve fibers have a stimulating or weakening effect on the Ia and II afferents. The γ -dynamic motor neurons cause an

increase in the phasic response and a decrease in the tonic response in the Ia-afferents. The γ -static motor neurons decrease the phasic and increase the tonic response of the Ia-afferent. With a constant γ -activity, the static response is proportional to the length of the muscle spindle and therefore proportional to the length of the muscle. Stretching of the muscle increases the unloading frequency while contraction decreases the unloading. Fast, short changes influence the Ia-afferents (phasic response) primarily. Large slow changes are primarily registered by the II-afferent (tonic response).

In vibration training, 100 percent of the muscles are involved and fast contractions occur. As a result, the unloading frequency of the Ia-afferents increases and the muscle contracts better due to the increased activity of the γ -dynamic motoneurons. The vibrations also inhibit the contraction of the antagonists through the Ia inhibiting neurons (Cardinale et al, 2003). Muscle spindles, therefore, function as important proprioceptors primarily in relationship with the joints and their related muscles. The actual function is to register the position of the joint and the speed and direction of the movement at the level of the joint, this is because the length of the muscle is determined by the position of the joint upon which the muscle acts. The temporal muscle displayed 342 (208 in the horizontal and 134 in the vertical portion), the masseter 114 (91 in the superficial and 23 in the deep portion), the medial pterygoid 59, and the lateral pterygoid muscle contained 6 muscle spindles. Unlike the spindles elsewhere in the body, spindles in the human jaw closers have been found to contain very large numbers of intrafusal fibers per spindle (up to 36) (Fig. 18.12). This finding reinforces the idea that the jaw-closer spindles should have a strong proprioceptive impact on the control of human mastication. Spindle discharge usually increases in animals when biting against an experimental load or hard food and hence, is correlated with the tension developed by their jaw muscles. The cell bodies of the afferents that connect these receptors to the central nervous system have been found to be located in the trigeminal mesencephalic nucleus.

- **Golgi-tendon organ:** The Golgi-tendon organs are located in the junction between the muscle and the tendon, as well as in the connective tissue surrounding

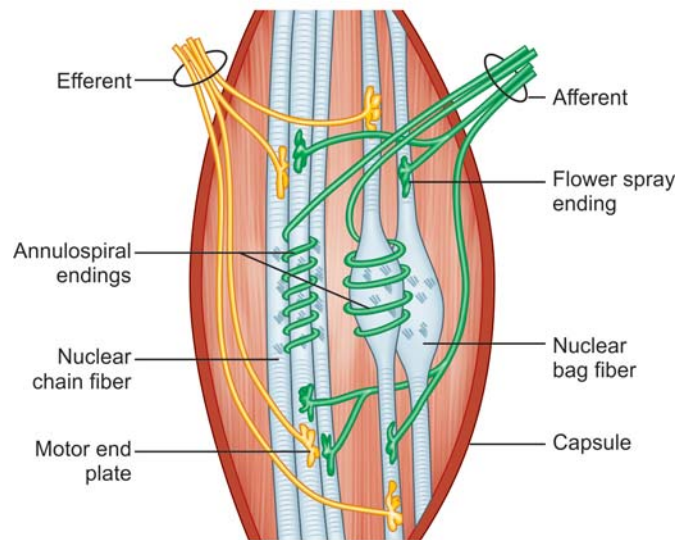


Fig. 18.12: Diagrammatic cross-section of a muscle spindle showing the intrafusal fibers; afferent and efferent endings

the joint. The Golgi-tendon organs run in series with the muscle and the tendon and therefore detect passive as well as active contractions. Contraction of one motor unit results in the excitation of at least one tendon sensor. The afferent fibers of the Golgi-tendon organs that connect to the central nervous system (CNS) provide information about the tension in the muscles. The Golgi-tendon organs, therefore function as tension detectors. The feedback provided is negative. This means that the Golgi-tendon organs inhibit the efferent nerves when muscle tension increases, resulting in a decrease of tension. There is only limited evidence of the existence of tendon organs in human.

- **Temporomandibular joint (TMJ) afferents:** Innervation of the human TMJ capsule and its receptor types have been studied. The major innervation of the joint comes from the auriculotemporal nerve (posterior and lateral portions). The anterior portion of the capsule receives innervations from the masseter nerves (Thilander, 1961). The articular surfaces of the joint and the meniscus, except for its peripheral border, are not innervated (Dubner et al, 1978). The receptor types found in the TMJ capsule include free nerve endings, Ruffini endings, Golgi organs, and Vater-Pacini corpuscles. It has been claimed that the Ruffini endings and the Golgi organ within the capsule

function as static mechanoreceptors, the Vater-Pacini endings as dynamic mechanoreceptors, and the free nerve endings as the pain receptors (Storey, 1976). The cell bodies of the afferents that connect these receptors to the central nervous system have been found to be located in the trigeminal ganglion.

- *Skin and mucosal receptors:* Human microneurography and psychophysical studies showed that other than the rapidly adapting receptors with large receptive area (type RAI; Pacinian endings), all other receptors exist in the human facial skin and mucosa (Barlow, 1987; Johansson et al, 1988). A majority of the mechanoreceptive afferent units in the skin of the human face are slowly adapting and have small and well-defined receptive fields (type SAI). Slowly adapting receptors with large receptive fields (SAII) and rapidly adapting receptors with small well-defined receptive fields (RAI) were also detected in these studies (Johansson et al, 1988).
- *Periodontal mechanoreceptors (PMRs):* Studies in the cat indicated that the majority of PMRs are located near the apex of the tooth root (Ness, 1954). Paring the alveolar bone overlying the root of the tooth and simultaneous mechanical and electrical stimulation were used to determine the properties of the adequate stimulus that initiated action potentials in these receptors (Linden, 1990). It was found that these receptors respond to tension but not to compression. With direct stimulation of the receptors, it has been found that the receptors with their cell bodies in the trigeminal mesencephalic nucleus are located in the middle of the fulcrum-apex, whereas the receptors whose cell bodies are situated in the trigeminal ganglion are distributed throughout the entire periodontal space. Directional sensitivity experiments have illustrated that the majority (over 70%) of the receptors with cell bodies in the trigeminal mesencephalic nucleus are located in the labial and mesial aspects of the teeth, whereas the receptors with cell bodies in the trigeminal ganglion are distributed more equally in the periodontium (reviewed in Linden, 1990). It has also been shown that the threshold of a particular receptor depended upon the rate of application of the force to the tooth crown. Immunohistochemistry (Maeda et al, 1990) study has shown that the periodontal space contains both free and specialized nerve endings. There are

four types of specialized nerve endings: Ruffini-like endings that are found mainly near the root apex; coiled nerve endings, found near the mid-range of the tooth root; and spindle and expanded nerve endings, both found near the root apex.

- *PMRs—muscle spindle interaction for calibrating the position of the jaw:* Central connections of the PMRs are quite unique in that most of these receptors have their cell bodies in the trigeminal mesencephalic nucleus along with the spindle cell bodies (Linden, 1990). It has been suggested that, in the trigeminal mesencephalic nucleus, an electrical link may exist between the cell bodies of spindles and periodontal receptors (Baker and Llinas, 1971; Taylor et al, 1978). It is also unique that the periodontal receptors and muscle spindles from jaw muscles have direct projections to the cerebellar cortex (Taylor and Elias, 1984; Donga and Dessem, 1993). It is thought that this direct connection can be used as a reliable signal of tooth contact, and this may be used to zero or recalibrate the spindle afferent discharges. Muscle spindles in the jaw-closing muscles give very finely graded information regarding mandibular movement. However, they cannot give reliable information about jaw position over a long period of time, because the spindle properties and the fusimotor activity change continuously during chewing (reviewed in Taylor, 1990). For the normal mandibular posture to be maintained, absolute positional information is needed, and that requires calibration of the muscle spindle afferent information with the exact time of tooth contact (Taylor and Elias, 1984). This calibration could be done by a comparison of the direct and reliable information received via the spindle and PMR afferents to the cerebellum (Taylor and Elias, 1984; Donga and Dessem, 1993). This comparison may allow the cerebellum to alter fusimotor activity appropriately and regulate the gain of the spindles in the jaw muscles (Prochazka, 1989).

Types of Reflex

Myotatic Reflex

It is the tonic contraction of the muscles in response to a stretching force, due to stimulation of muscle proprioceptors. It is also called as Liddell-Sherrington reflex, muscular reflex, and stretch reflex.

The *stretch reflex* requires sensory neurons that supply muscle spindles and motor neurons that supply the extrafusal fibers of the muscle. This reflex is ordinarily suppressed by the activity of descending pathways that end on motor neurons and (especially) on nearby interneurons. It can be elicited by suddenly lengthening a muscle, as when the tendon is tapped (jerk reflexes). A myotatic reflex occurs when a passive stretch of a muscle causes the muscle to contract. Sufficient γ -activity during the stretching of the muscle and muscle spindles can result in the excitation of the Ia-afferents. These in turn, stimulate the α -motoneurons in the spine causing the extra-fusal fibers of the muscle to contract. Under normal circumstances the antagonists will relax.

Mechanism of stretch reflex: (Fig. 18.13) The stimulus for stretch reflex is the stretch of the muscle. Muscle spindles act as stretch receptors. When the stretch receptors are activated, it causes contraction of the stretched muscle. Muscle spindles are located within the muscle itself and it is made up of 2 to 15 thin intrafusal fibers. The slender ends of the intrafusal fibers are striated and contractile while the central or nuclear bag is non-contractile. Impulses arising from the muscle spindle are conducted by the group IA sensory nerve fibers. These sensory nerve fibers synapse with the motor neuron known as alpha efferents that supplies the extrafusal muscle fibers responsible for the contraction of the

stretched muscle. The myotatic reflex is a type of monosynaptic arc. The stretch reflex serves as a mechanism for upright posture or standing. In the mandible, the stretch reflex acts to maintain the postural rest position of the mandible in relation to the maxilla.

Regulation of myotatic reflex: Higher centers of brain control the myotatic reflex through the reticular formation. Apart from the alpha efferents supplying the extrafusal fibers of the muscles, smaller motor neurons or gamma efferents supply the intrafusal fibers of the muscle spindle. Activation of the gamma efferents will cause polar contraction of the intrafusal fibers and therefore puts the noncontractile nuclear bag under tension. This causes a mechanical distortion which is similar to passive stretch of the muscle. So gamma efferents will initiate spindle discharge and increase the sensitivity of the spindle and act like a blasting mechanism regulating the sensitivity of muscle spindles. Through these gamma efferents, the higher centers of the brain via reticular formation influence the stretch or myotatic reflex. The reticular formation influences the myotatic reflex by facilitation or inhibition of the small gamma efferents which cause contraction of the intrafusal fibers of the muscle spindles thereby increasing the rate of the spindle firing which in turn influences the amount of alpha motor neuron firing.

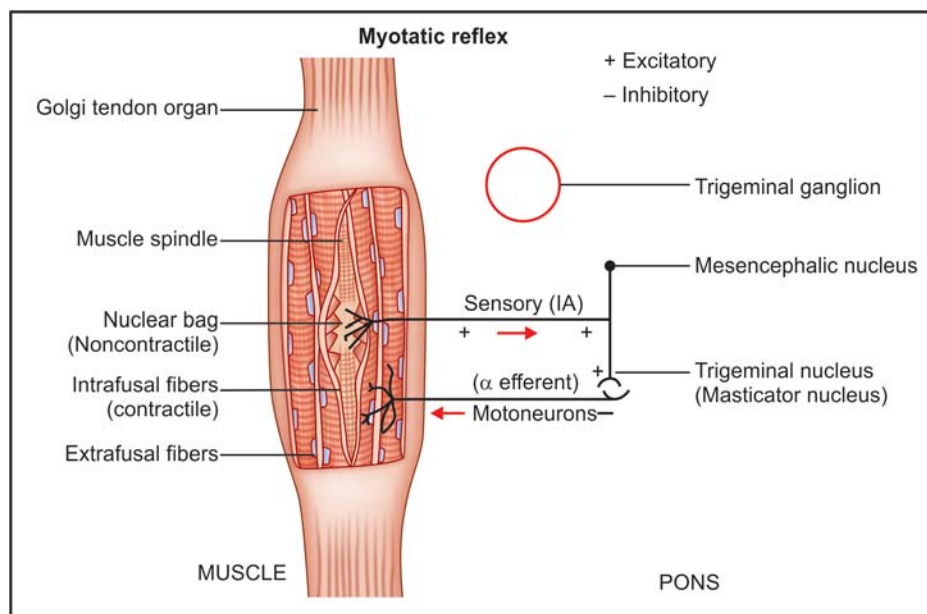


Fig. 18.13: Diagrammatic representation of myotatic reflex

Clasp Knife Reflex

This phenomenon, sometimes called a reflex, is produced by stretching an extensor muscle against a background of increased extensor muscle tone. The result is a relaxation of the muscle being stretched, i.e. the muscle now lengthens easily after initial resistance. The "reflex" is assessed by passively flexing a patient's limb, at the knee or elbow, an action that passively extends the extensor muscles. Clasp knife reflex is also called as autogenic inhibition or inverse myotatic reflex.

Mechanism (Fig. 18.14): Muscle first resists, then relaxes. This resembles that of a spring-loaded folding knife blade and hence, this phenomenon is called the "clasp-knife" reaction. The excessive or rapid stretch of the muscle brings in to play some inference that annuls the stretch reflex and allows the muscle to be lengthened with little or no tonic resistance. Thus, the stimulus necessary to elicit the clasp knife reflex is excessive stretch and when elicited, it inhibits muscular contraction, thus causing the muscle to relax. The receptors for the clasp knife reflex are the Golgi tendon organs located in the tendon of the muscle. The impulses are conducted by group 1B

sensory nerve fibers, the impulses act on the motor neuron of alpha efferent supplying the stretched muscle. However, it is a di-synaptic reflex arc because an interneuron is interposed between the sensory neuron and the motor neuron. It follows during the muscle stretch; the motor neurons supplying the stretched muscles are bombarded by impulses delivered over two competing pathways, one facilitating and other, inhibiting muscle contraction. The output of the motor neuron pool depends upon the balance between the two antagonists inputs. The functional significance of the clasp knife reflex is to protect the overload by preventing damaging contraction against strong stretching forces. In the inverse myotatic reflex, the Golgi tendon organs are more strongly activated when they detect that too much weight is being put on a muscle.

Jaw Closing Reflex

Jaw closing reflex is the most basic reflex in the facial and oropharyngeal area. Jaw closing reflex is sometimes referred to as jaw jerk reflex.

The structure of this reflex, with the components discussed above, is shown in Table 18.2.

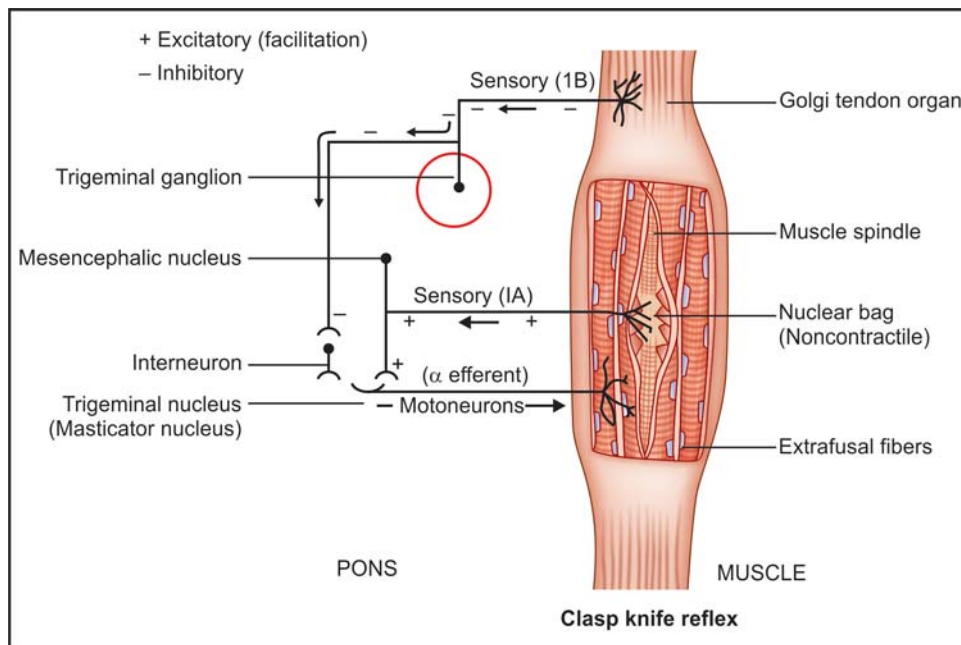


Fig. 18.14: Diagrammatic representation of clasp knife reflex. During initial stretching of muscle, resistance is felt due to hyperactive reflex contraction of the muscle in response to stretch. Further stretching will cause the resistance to disappear and collapse of the rigid muscle takes place like a spring loaded knife. Clinical significance includes not advancing the mandible to a very great extent during construction bite so that the Golgi tendons are not activated

Table 18.2: Components of jaw closing reflex

Receptor	Muscle spindle in jaw closer muscles, PDL receptors, etc.
Primary afferent	Spindle Ia afferent
Cell body	Mesencephalic nucleus of V nerve
Central process	Monosynaptic connection with trigeminal motoneurons
Motoneurons	Jaw closer motoneurons in motor nucleus of V nerve
Appropriate stimulus	Opening of jaw, stretching of jaw-closer muscles
Response	Contraction of jaw closer muscles; jaw closing
Function	Maintain jaw position against gravity and inertial loading

The mandible is maintained in rest position by the activity of the jaw-closer muscles, mediated through the stretch reflex. When we fall asleep in a sitting position, the neural control of jaw position is disturbed, with the result that the jaw droops and the mouth may fall open. When we are awake and jump or run, the jaw is kept closed by the jaw closing reflex; the stretch on the muscle spindles caused by bouncing up and down causes the jaw-closing muscles to stiffen at the right time to keep the jaw elevated. An additional jaw closing reflex during biting will be described later. The jaw-closers, which work against gravity for most of the day, are rich in type I fatigue-resistant muscle fibers.

The jaw-jerk, obtained when a sharp blow is delivered to the chin, is diagrammed in Figure 18.15. Like the knee jerk, it is mediated through the monosynaptic stretch reflex, in this case in the brainstem. A sudden tap on the chin lengthens the muscle spindles and causes a fairly synchronous activation of the closer muscles. The latency of the response, from stimulus to contraction, is about 10 ms. Pathway 1 is primarily excitatory and pathway 2 is inhibitor to jaw closing motoneurons. The pathway through V mesencephalic nucleus to trigeminal motor nucleus is direct but pathway 2 through V ganglion involve one or two synapses. Some of the afferent information may pass to higher centers (cerebral cortex).

Jaw Opening Reflex

This reflex is the first reflex movement to make its appearance in the orofacial region of human beings at about 8.5 weeks of intrauterine life. The general structure of the jaw opening reflex is shown in Table 18.3. This is sometimes known as the linguomandibular reflex, since it also occurs with brief application of a noxious stimulus to the tongue.

When we bite down on a hard object, enormous forces can be generated by the jaw closer muscles (from 50-150 kg), which is more than enough to break a cusp off of a tooth. To prevent this type of injury from excess forces on the teeth, there are mechanoreceptors in the

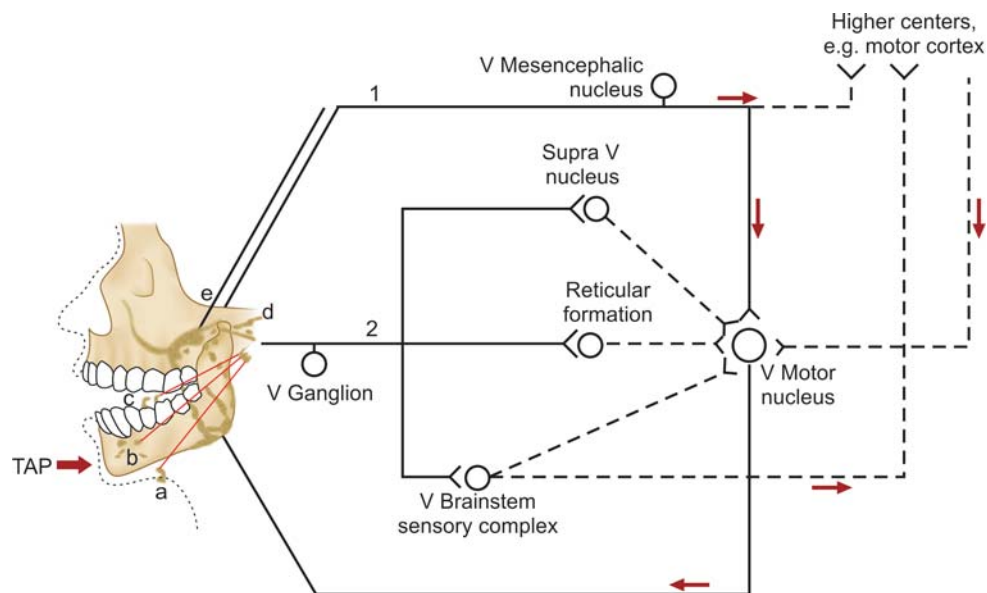
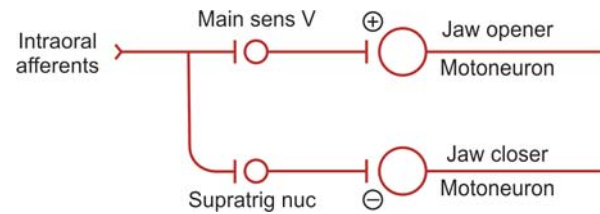


Fig. 18.15: Possible receptor sites (a. cutaneous; b. periodontal; c. mucosal; d. TMJ; e. muscle spindle) and central reflex pathways (1 and 2) involved in jaw closing following a tap in the region of chin

Table 18.3: Components of jaw opening reflex

Receptor	Mechanoreceptors and pain receptors in oral cavity.
Primary afferent	Trigeminal sensory axons.
Cell body	Trigeminal ganglion.
Central process	Synapse with interneurons in sensory nucleus of V, which synapse with motoneurons.
Motoneurons	In humans, closer motoneurons are inhibited.
Appropriate stimulus	Short-onset, high-intensity localized mechanical or noxious stimulus within the oral cavity.
Response	Inhibition of jaw closers; jaw opening.
Function	Protection of hard and soft tissues of oral cavity.

periodontal ligament which are sensitive to tooth displacement. Any tooth contact during jaw closing immediately inhibits the closing muscles. Likewise, the soft tissues such as the tongue, cheeks and lips may easily be injured by the teeth, for instance during mastication. Pain receptors in the soft tissues are also able to stop the jaw from closing within about 15 ms of biting the tissues. The afferents from these receptors are connected as in Figure 18.16. The graphical summary of the neural connections of the oral reflexes is shown in Figure 18.17

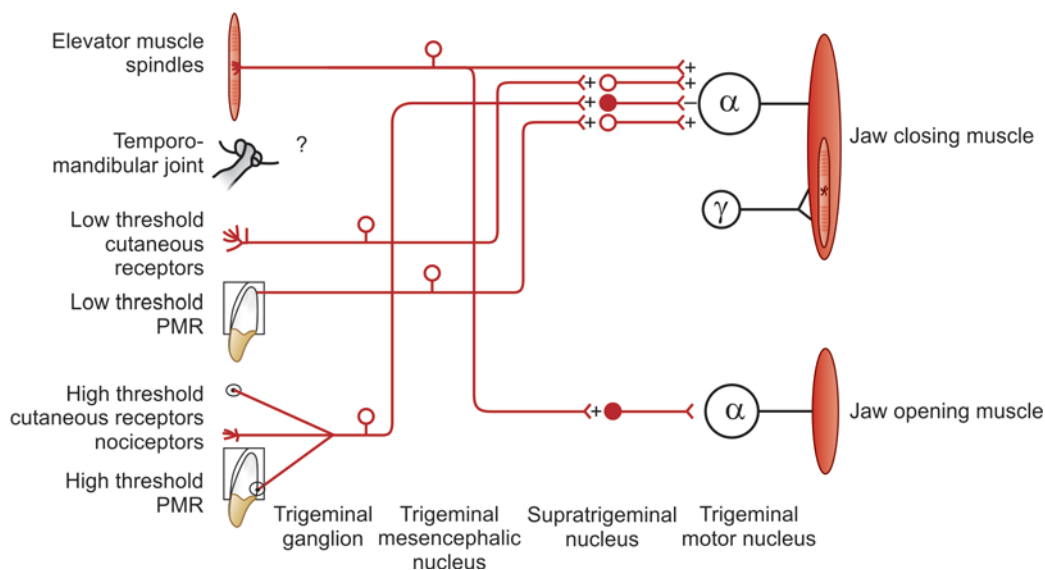
**Fig. 18.16:** Connections of jaw opening reflex

(from Türker, 2002). Sensory endings are shown on the left, sensory and motor nuclei in the center and effector muscles on the right.

Antigravity and protective jaw reflexes thus act constantly to assist with various oral functions. Studying the reflexes has helped neuroscientists to understand the underlying neural connections.

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**Fig. 18.17:** Neural connections of oral reflex

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Maturation of Orofacial Functions

CHAPTER OUTLINE

- **Respiration**
- **Swallowing or Deglutition**
 - Infantile swallow
 - Mature swallow
 - Stages of swallowing
- **Mastication**
 - Neurological control
 - Child and adult chewing patterns
 - Murphy's six strokes of mastication
- **Speech**
 - Neurophysiology
 - Mechanism of speech production
 - Subsystems of speech
 - Maturation and description of speech
 - Consonants and vowels
 - Milestones of speech development

The term 'maturation' literally means the process of becoming mature. It denotes the emergence of personal, behavioral and physiological characteristics through growth process. Orofacial musculature and functions are relatively the most sophisticated in the new born to ensure that the patency of airway is maintained and nutritional demands are met.

The important physiologic orofacial functions include respiration, swallowing, mastication, and speech. The process of maturation of each of the orofacial functions can be studied under three stages:

1. Prenatal orofacial function.
2. Neonatal orofacial function.
3. Postnatal development of orofacial functions.

Most of the orofacial reflexes present are unconditioned reflexes as they are essential for survival of life. The unconditioned reflex includes jaw opening

reflex, respiration, suckling and swallowing, gag reflex. Table 19.1 shows the time of development of different reflexes during intrauterine life. In the new born infants, mouth is the only area of communication with the external world and sensory development of the oral cavity is highly developed when compared to the other areas of the body. Orofacial functional maturation occurs prior to trunk and limb regions. This is highly essential as mouth is the primary site for life sustaining reflexes like respiration, feeding and airway maintenance.

The orofacial functional maturation takes place by way of postnatal development of the pharynx and the mouth. In early infancy, these two organs perform as a closely knit composite. In this integration the pharynx, the more primitive element, is commonly dominant. Postnatally, the mouth acquires autonomy and differentially progresses to perform the most complex and heterogeneous actions effected by our motor mechanisms. These are the results of developmental encephalization, whereby the successively acquired representations of the mouth are integrated with the

Table 19.1: Tentative time schedule for initiation of orofacial reflexes

Type of reflex	Time of commencement (IUL)
Jaw opening reflex	8.5 weeks
Lip stimulation causing tongue to move	14 weeks
Gag reflex	18.5 weeks
Respiration	25 weeks
Suckle	29 weeks
Suckling and swallowing	32 weeks

maturing environmental orientation, intelligence, and emotions of the organism. The maturation of orofacial function follows a front to back maturation. At the time of birth, lips are mature and perform vigorous activity, while the posterior structures are immature.

RESPIRATION

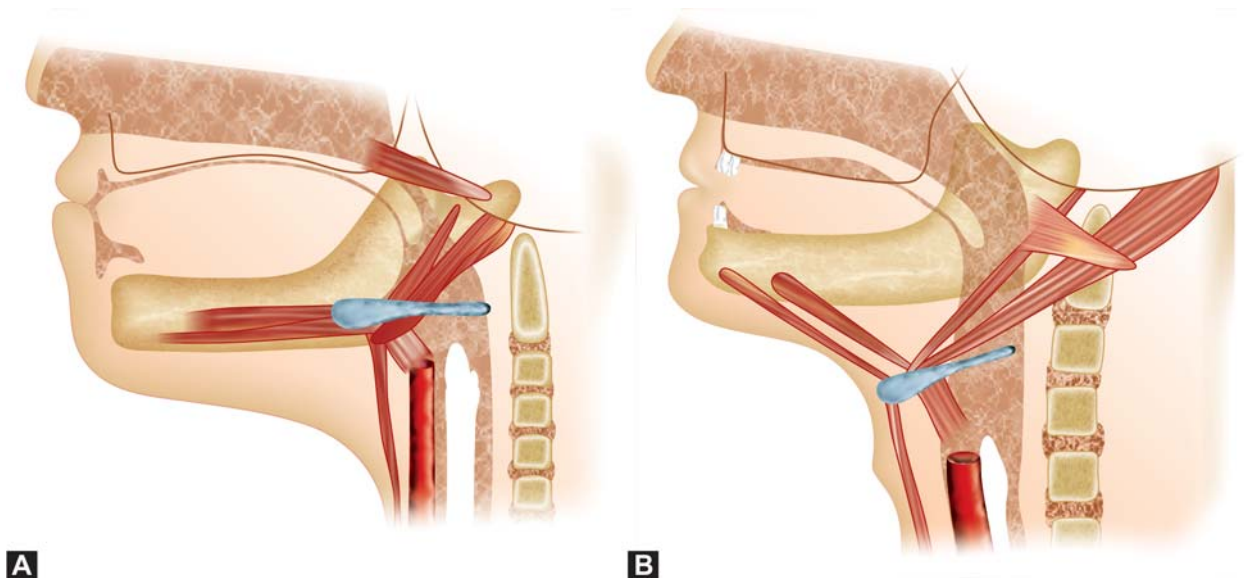
Physiologic respiration is the process by which multicellular living organisms capture and excrete the gaseous fuel and waste of cellular respiration. During this process oxygen is absorbed and carbon dioxide is disposed. Even though nose forms the primary portal of entry for respiration, the mandibular and tongue posture are major determinants of respiration.

Respiration starts at about 25 weeks of intrauterine life. But at this stage the lungs do not inflate. James Bosma (1963) in his classic study has shown that the mandible moves downward and tongue also moves downward and the forward from the posterior pharyngeal wall to establish the airway. This act by the child permits the passage of air through nose and pharynx into the lungs. Neonates are obligate nasal breathers and if the nasal airway is blocked, their survival becomes difficult. Later, breathing through the mouth becomes possible for the child. Positional stabilization of the dorsal portion of the mouth is a function shared with the pharynx and is also a part of pharyngeal

participation in respiration (Bosma, 1969). Throughout postnatal life, the column arrangement of tongue, hyoid, and larynx is held forward, maintaining patency of the airway in the pharynx and the laryngeal vestibule. This column is also held upward, with the tongue in approximation to the palate, so that the airway is in continuity with the nose, rather than the mouth. The soft palate is active in this approximation, separating the mouth from the pharynx (Fig. 19.1A). With the postnatal elongation of the vertical array of the mandible, hyoid, and pharynx, the composition of the anterior wall of the larynx is changed by the appearance of the tongue between the palate and the epiglottis (Fig. 19.1B).

Mouth and nose forms the anatomic beginning of the respiratory system. Patency of the airway in the nose and oral cavity is maintained by the bony skeleton and adaptive posture of the tongue. In the pharynx, the patency is again dependent upon the tone of the muscles of tongue, soft palate and pharyngeal walls.

The larynx lies at the level of upper cervical vertebrae C4-C6. The main structural components of larynx are the thyroid and cricoid cartilages, along with smaller arytenoid cartilages and the epiglottis which sits over the laryngeal inlet. The miraculously efficient, split second opening and closing of the epiglottis, preventing the food from entering the airway is a wonder of nature. Development of respiratory spaces and maintenance of



Figs 19.1A and B: Pattern of spatial orientation of mouth and pharynx and of hyoid suspensory muscles in the newborn infant (A) and in the adult (B). AJO-DO, 1969 Jun (32-38): Evaluation of oral function of the orthodontic patient—Bosma

oral and pharyngeal airway space contribute to the growth of orofacial bones according to functional matrix hypothesis.

Clinicians and researchers involved in the treatment of dentofacial deformities have long searched for determinants of facial morphology. Since the turn of the century, nasal airway function has been implicated as an etiologic factor in dentofacial development. *First theory* proposing the existence of a relationship between mouth breathing and facial form stated that oral respiration alters normal air currents and pressures through the nasal and oral cavities, which causes impaired development of these structures. Several authors (Morrison and Bloch) postulated this to be the result of the oral airstreams in mouth-breathing individuals hindering normal downward palatal growth. Others like Angle believed that the raised negative air pressure difference between the oral and nasal passages in mouth breathers led to development of a deep palatal vault.

A *second theory* held that oral respiration disrupts the muscle forces exerted by the tongue, cheeks, and lips upon the maxillary arch. The mouth breather was believed to position the tongue in a more downward and forward manner in the oral cavity, a position in which it could not exert adequate buccal pressure to counteract the inward forces from the lips and cheeks upon the maxilla (Harvold, Linder-Aaronson et al). This theory called the compression theory exists in current literature.

A *third school* of thought denies a significant relationship between facial morphology and mode of breathing. Kingsley was among the first to consider the V-shaped maxillary arch and deep palate a congenital trait not related to mouth breathing. In a subjective evaluation of 1,033 children, Humphrey and Leighton reported an approximately equal distribution of malocclusions in nose and mouth breathers. They noted that, of those children who kept their mouths open while breathing, almost half respired nasally. Gwynne-Evans and Ballard also subjectively evaluated the relationship between facial morphology and breathing conditions over a period of 15 years. They reported that orofacial morphology remains constant during growth, regardless of breathing patterns. They also stated that "mouth breathing does not produce deformities of the jaws and malocclusions and does not result in the development of the adenoidal facies". Leech examined the relationship

between lip competence and mode of breathing in subjects undergoing evaluation in a research clinic for upper respiratory disease and found that less than one third of the lip-incompetent persons were mouth breathers. Thus, many theories have been proposed and much confusion remains regarding the relationship between nasorespiratory function and dentofacial morphology.

SWALLOWING OR DEGLUTITION

Deglutition is the act or process of swallowing. Once the respiration is established in an infant, the next important event or priority is suckling and swallowing. These two maneuvers help the child to obtain milk and transfer it to the gastrointestinal tract. Both suckling and swallowing movements start developing from 32nd week of intrauterine life.

Suckling consists of small nibbling movements of the lips around the mother's breast to stimulate the smooth muscle contraction which causes the squirting of milk into the mouth. Thus the suckling maneuver is entirely different from the sucking process. Once the milk is squirted into the mouth, the neonate or infant positions the tongue anteriorly in such a way, the tongue is in contact with the lower lip. This facilitates the deposition of milk on the tongue. Once deposited, the infant grooves the tongue so that the milk flows posteriorly into the pharynx and esophagus.

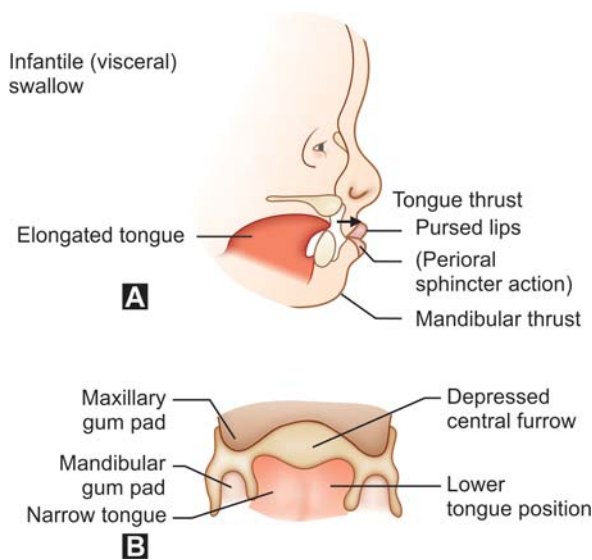
Infantile Swallow (Figs 19.2A and B)

Suckling is associated with the type of infantile swallowing mechanism. It is also called as visceral swallow. Fletcher stated that the infantile swallow is because of the significant difference in size or morphology of the oral cavity and the increased tongue size. Moyers listed the features of infantile swallow as follows:

1. Jaws are apart with the tongue interposed between the gum pads.
2. Mandible is stabilized by the contraction of muscles of facial expression and by the interposed tongue.
3. Swallowing is guided and controlled by the sensory interchange between the lips and tongue.

Mature Swallow

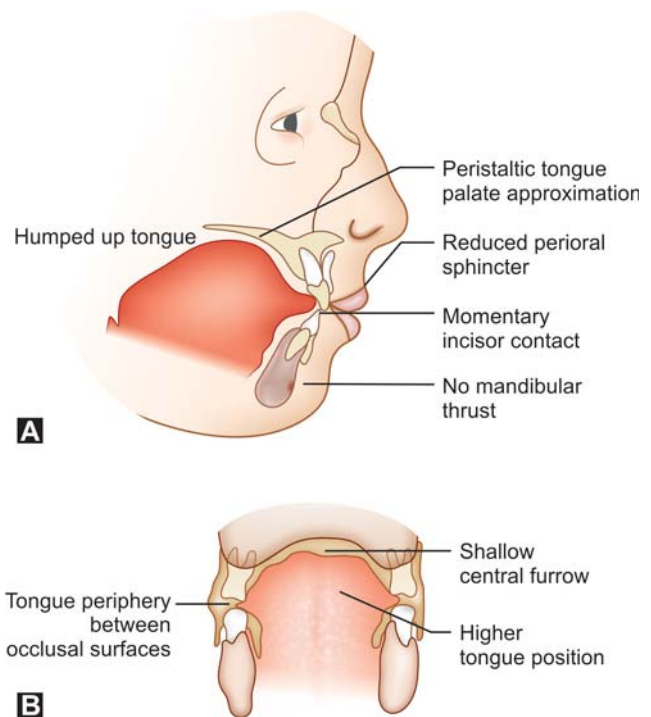
Subsequent to the eruption of teeth and shift to semisolid and solid food, the infantile swallow disappears during



Figs 19.2A and B: Infantile swallowing mechanism. (A) Profile view and (B) Posterior view Plunger-like action is associated with nursing. Cheek pads flow between posterior gum pads during nursing, unopposed by peripheral portions of tongue. Associated with the tongue-thrust is the anterior positioning of the mandible. The condyle may be felt gliding rhythmically forward and backward in the nursing act. (AJO, Note concave midline contour of dorsum of tongue. 1963 Jun (418-450): The "three M's": Muscles, Malformation and Malocclusion—Graber)

the end of first year of life. At about 5 to 6 months of age, as the incisors begin to erupt, certain proprioceptive impulses come into play and the peripheral portion of the tongue starts to spread laterally. This change in tongue function is a gradual one, and it is called the transitional stage. As the incisors erupt completely, the peripheral portions of the tongue occupy the space between the remaining edentulous areas of the upper and lower gum pads, and the more mature somatic swallow is the result (Figs 19.3A and B). The lips close, and the incisors come together momentarily as the tip of the tongue lies behind the incisors during the swallowing act. By 18 months of age, the mature swallowing pattern is usually observed in a child.

Tongue is no longer placed between the gum pads or incisors. There is diminishing of anterior mandibular thrust, the muscles of mastication take control of the position of mandible, the tip of tongue is retracted and placed behind the incisive foramen. There is no or only minimal contractions of lips during mature swallow and the teeth are held together.



Figs 19.3A and B: Mature swallowing mechanism (A) Profile view and (B) Posterior view. The dorsum is less concave and approximates the palate during deglutition. The tip of the tongue is contained behind the incisors; peripheral portions flow between opposing posterior segments. Anterior mandibular thrust has disappeared. (AJO, 1963 Jun (418-450): The "three M's": Muscles, Malformation and Malocclusion—Graber)

Swallowing is a complex neuromuscular activity involving rapid coordination of structures in the oral cavity, pharynx, larynx, and esophagus. These structures must also support the physiologies of respiration, phonation, and articulation, in addition to deglutition. In normal adults, respiration ceases during the process of deglutition since the food bolus crosses the pathway that air takes on its way to the lungs. There are three important prerequisites for mature swallow. They are (i) establishment of pressure gradient; (ii) prevention of reflux; and (iii) protection of airway. Obligate muscles are the group of muscles which function for these requirements to be fulfilled. Table 19.2 gives details of the requirements of swallowing.

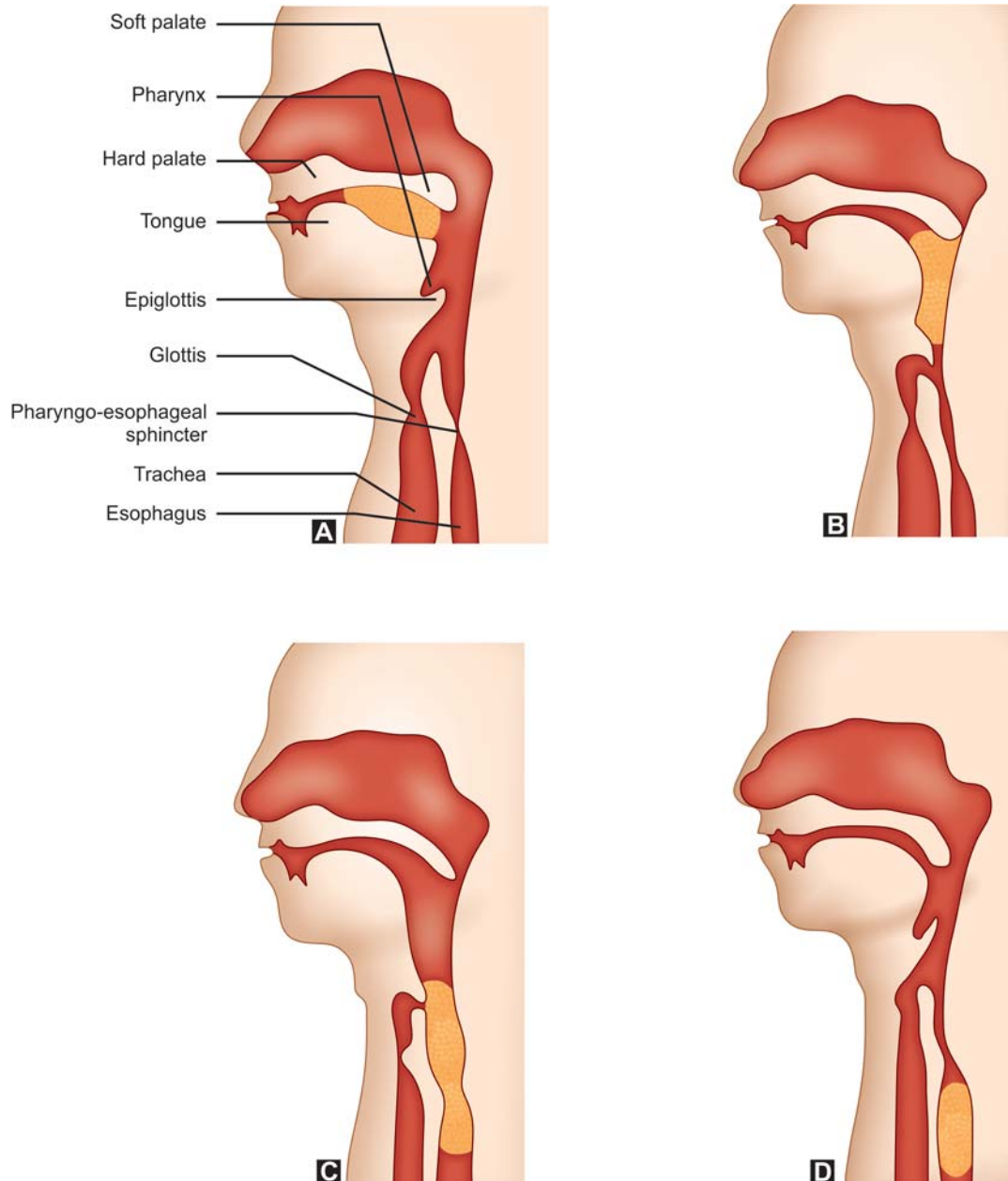
Stages of Swallowing (Figs 19.4A to D)

Normal swallowing can be divided into four stages:

1. Oral preparation stage.
2. Oral stage.

Table 19.2: Essential requirements of normal mature swallow

Pressure gradient	Muscles involved	Prevention of reflux	Airway protection
• Tongue piston action • Tongue base stabilization • Pharyngeal constrictors stripping action • Esophagus: peristalsis	Floor of mouth, facial expression, elevator muscles	• Anterior oral seal by lips, incisors and tongue • Tongue palate apposition • Hypopharyngeal sphincter • Gastroesophageal sphincter	• Palate and pharyngeal wall apposition • Larynx elevation • Vocal folds adduction • Apnea



Figs 19.4A to D: Four stages of deglutition: (A) Preparation stage, (B) Oral stage, (C) Pharyngeal stage, (D) Esophageal stage

3. Pharyngeal stage.
4. Esophageal stage.

The first two stages are under voluntary control, whereas the second two stages are involuntary, being under reflexive control. Cortical control of swallowing is in the anterolateral cortex. Fibers descend through the internal capsule to the substantia nigra and then to the mesencephalic reticular formation. The brainstem “swallowing center” is thought to be in the medulla between the posterior pole of the facial nucleus and the rostral pole of the inferior olive. On an average an individual swallows about once a minute between meals and 9 times a minute during eating. Swallowing is present even during sleep.

1. Oral Preparation Stage

A. Purpose:

1. Solid food reduced mechanically by mastication is mixed with saliva to produce a pulverized consistency appropriate for swallowing.
2. Produces the pleasurable sensation of eating.

B. Neuromuscular actions:

1. Lip closure to hold food in the mouth anteriorly.
2. Tension in the labial and buccal musculature to close the anterior and lateral sulci.
3. Rotary motion of the jaw for chewing.
4. Lateral rolling motion of the tongue to position food on the teeth during mastication.
5. Bulging forward of the soft palate to seal the oral cavity posteriorly and widen the nasal airway.

- #### C. The most important neuromuscular action of this stage is the tongue motion since it is extensively involved in the manipulation and mastication of food, as well as the formation of the food into a cohesive ball at the end of this stage. It does so by cradling the bolus by central depression and lateral elevation, positioning it against the palate in a cohesive mass.

2. Oral Stage

A. Purpose:

Move food from the front of the oral cavity to the anterior faucial arches, where the reflexive swallow is initiated.

B. Neuromuscular actions:

1. Tongue makes vertical contact anteriorly with alveolar ridge.

2. Vertical tongue-to-palate contact progresses posteriorly, propelling the bolus ahead of it toward the pharynx.

- #### C. As in oral preparative stage, tongue motion is most important. Requires fine muscular control of tongue to elevate and move in a smooth anterior to posterior direction. This stage lasts less than one second in duration. Innervation is primarily from the vagus in the brain stem, but involves cranial nerves IX through XI.

3. Pharyngeal Stage

A. Purpose:

1. Transport food from the faucial arches to the esophagus.
2. Protect the airway by preventing aspiration.

B. Neuromuscular actions:

1. Velopharyngeal closure, to prevent entry of food or liquid into the nasal cavity by:
 - a. Velar elevation by levator veli palatini and tensor veli palatini.
 - b. Velar retraction by palatopharyngeus muscle.
 - c. Anterior movement of posterior pharyngeal wall by superior pharyngeal constrictor—Passavant's pad.
 - d. Medial movement of lateral pharyngeal walls (superior constrictor).
2. Pharyngeal peristalsis, to propel bolus through pharynx and clear food residue from the pharyngeal recesses such as the valleculae and pyriform sinuses. Begins after tongue base retraction which drives the bolus at first. Bolus then pushed through pharynx by sequential contraction of the superior, middle, and inferior constrictors.
3. Airway protection to prevent aspiration through elevation and closure of larynx:
 - a. Larynx closes at three different levels during swallowing to prevent aspiration. True vocal cords close first, followed by the false vocal folds, and then by the approximation of the aryepiglottic folds and the coverage of the superior laryngeal inlet by the epiglottis.
 - b. Hyoid is suspended in the neck by the suprahyoid muscles (ant. digastric, mylohyoid, geniohyoid anteriorly; stylohyoid and post.

digastric posteriorly). Larynx is suspended in neck by muscle (thyrohyoid) and ligaments attached to hyoid. Hyolaryngeal complex is elevated and moved forward during this stage. These actions tuck the larynx under the tongue and floor of mouth, up and out of the way of the passage of the food bolus.

- c. Anterior, forward movement of hyolaryngeal complex is the most important in preventing aspiration, followed in importance by glottic closure.

- 4. Opening of the cricopharyngeal region allows bolus passage into the esophagus. Upper esophageal sphincter (UES) is made up of cricopharyngeus attached to cricoid cartilage. At rest, UES is closed to prevent air intake into the esophagus during breathing and to prevent reflux from the esophagus into the pharynx. Exact timing or triggering mechanism is unknown, but several factors contribute to opening of UES: (a) relaxation of UES to enable opening (b) upward, forward movement of larynx which is believed to be most important in opening the cricopharyngeal sphincter. (c) bolus pressure increases the width of UES opening.

- 5. Tongue base retraction over bolus and pharyngeal contraction also aid in propulsion of food through the pharynx.

C. Lasts less than one second.

4. Esophageal Stage

A. Purpose:

- 1. To transport the food bolus from the UES to the stomach.

B. Neuromuscular actions:

- 1. Extends from level of cricoid cartilage (C6) to the cardia of the stomach with average length of 25 cm in males and 23 cm in females.
- 2. During the esophageal stage, there is generation of “primary wave” with a force of 100 cm water pressure which moves the bolus along the length of the esophagus.
- 3. “Secondary wave” can be generated when there is increased pressure in mid-esophagus as occurs with residual food left in the esophagus after the completion of the “primary wave”.

- 4. “Tertiary wave” may occur in the elderly and in certain pathologic states. Occurs in the distal esophagus and makes a non-progressive, corkscrew-like motion.

- 5. UES opens with only 25 cm water pressure from above, but greater than 100 cm water pressure is needed to open it from below. LES opens with 5 to 7 cm water pressure from above, but greater than 25 cm water pressure is required to cause reflux.

- C. Esophageal stage lasts 8 to 20 seconds. Note that 90 percent of the swallow occurs during expiration; an apneal pause between 1 and 3.5 seconds in duration occurs during the oral and pharyngeal stages (Logemann, 1989; 1997).

MASTICATION

Mastication is defined as *the reduction of food in size, changing in consistency, mixing it with saliva and forming into a bolus suitable for swallowing*. Mastication is the action of breaking down of food, preparatory to deglutition. This breaking-down action is a highly organized complex of neuromuscular and digestive activities that, in normal individuals, integrate the various components of the masticatory system, such as the teeth and their investing structures, the muscles, the temporomandibular joints, the lips, the cheeks, the palate, the tongue, and the salivary secretions.

The object of chewing is to crush, triturate and mix food with saliva, so that food can be transported by deglutition down the digestive canal. The most important muscles for this purpose are temporal (anterior and posterior), the masseter (superficial and deep), the medial pterygoid, the lateral pterygoid (superior and inferior), and the digastric muscles. The trigeminal motor nucleus of motoneurons innervating the jaw muscles lies across the midline of the brainstem. However, mastication involves far more muscles than these “muscles of mastication” innervated by the trigeminal nerve. Synergistic movements of muscles innervated by facial and hypoglossal nerves are equally important.

The masticatory sequence is the whole set of movements from ingestion to swallowing. It is made up of masticatory cycles that change in form as the food is gathered, moved backward to the molar teeth, then broken down and prepared for swallowing. It is possible

to distinguish between cycles which occur at the beginning of the masticatory sequence and form the preparatory series of movements, cycles of particle reduction and cycles related to preswallowing (Lund JP). The cycles of reduction are intermediate in duration, longer than the preparatory cycles, but shorter than the preswallowing ones.

Neurological Control of Mastication

Jaw movements are among the most complex and unique movements performed by the human body. The mandible, unlike any other bones in the human body, is slung between two nearly symmetrical joints, which are close to being the mirror image of one another. Each muscle involved in the control of mastication has its counterpart on the opposite side of the jaw (Neeman et al). To create precise mandibular movements, inputs from various sensory receptors must be received by the central nervous system through afferent nerve fibers. The brain assimilates and organizes these inputs and elicits appropriate motor activities through the efferent nerve fibers. These motor activities involve the contraction of some muscle groups and the inhibition of others. Chewing is a subconscious activity, yet can be brought to conscious control at any time (Okeson). The coordination and rhythmicity of mastication has been attributed to the alternate activation of two simple brain stem reflexes. These are the *jaw-opening reflex*, activated by tooth pressure or tactile stimulation of wide areas of the mouth and lips, and the *jaw-closing reflex*, which follows stretching of the elevator muscles during opening (Sherrington and King et al). The introduction of a food bolus into the mouth was thought to initiate a self-perpetuating cycle by producing jaw opening, and the consequent stretching of the elevator muscles would produce jaw closure on the bolus, again producing jaw opening by stimulation of periodontal and soft tissue receptors (Dellow and Lund). The same authors found that in rabbits the timing of rhythmic chewing occurs within the brainstem. They suggested that mastication is controlled by a pattern generator brought about by reverberating circuits within the brainstem and that this patterning can be activated by adequate inputs from higher centers or from feedback through sensors in the oral cavity. The control of mastication is dependent in large part on the sensory feedback, which consists of epithelial mechanoreceptor afferents, periodontal

afferents, temporomandibular joint afferents and muscle afferents. Sensory feedback may explain the coordination of the tongue, lips, and jaws to move the food around, the reason why different foodstuffs influence the pattern of masticatory movement or the abrupt changes from cycle to cycle. While the cortex is the main determiner of action, centers in the brain-stem maintain homeostasis and control normally subconscious body functions. Within the brain-stem is a pool of neurons—a central pattern generator (CPG)—that controls rhythmic muscle activities. The neurons can be activated by adequate inputs from higher centers or from the oral cavity, and it is responsible for the precise timing of activity between synergetic and antagonistic muscles, so that specific functions can be carried out. Sensory feedback interacts with the control system at several levels to adapt the rhythmic program to characteristics of the food. This feedback is also a source of the variability in masticatory movements. Once an efficient chewing pattern is found, it is learned and repeated. This learned pattern is called a muscle engram. Chewing therefore can be thought of as an extremely complex reflex activity. The brain-stem also contains other areas, such as the reticular system, the limbic system and the hypothalamus, that have influence on masticatory function. These structures can modify the response of the cortex to any given stimulus, modify motor neuron activity, and even initiate irrelevant muscle activity. Thus, features of mastication can be programmed by the brain stem in the absence of sensory inputs, but such movements would be highly inefficient and even dangerous to the masticatory system. For a detailed study about jaw opening and closing reflexes, refer to chapter on muscle and receptors.

Child and Adult Chewing Patterns

The chewing pattern of the child is different from that of an adult. Development of mastication in a child requires development of new sensory motor patterns. The most important factor in the maturation of mastication is the sensory aspect of eruption of teeth. Moyers in 1964 has stated after serial EMG studies that the jaw muscles begin to learn mastication process when the maxillary and mandibular incisors touch one another. The first or earlier chewing patterns are poorly developed and the chewing pattern in a child gets stabilized when the complete primary dentition is erupted. Both jaw opening and closing reflex come into play but the role of condylar

guidance is not significant at this stage. Typically the child chewing pattern is as follows: child moves the jaw first laterally on opening and then the masticatory cycle is performed.

The chewing pattern in an adult is as follows: First an adult opens straight down, moves the jaw laterally and then brings about teeth contact. The transition to adult chewing pattern occurs during the eruption of permanent canines (about 12 years of age). The individual's movements during chewing are the result of integrated pattern of different functional components. It includes three systems, namely bone, teeth and muscle. Masticatory frequency appears to be one to two strokes per second with normal bolus of food. It can also vary in different conditions.

Chewing must be learned, and occurs only after tooth eruption. It is possible that periodontal ligament receptors and their stimulation are essential for this learning process. The masticatory envelope is usually described as a “tear-drop shaped” with a slight displacement at the beginning of the opening phase. This means that the opening movement rarely goes straight down. In most cases it deviates to the chewing side. The maximum extent of vertical and lateral movement in normal mastication is about half of the maximum vertical and lateral movement possible. Figure 19.5 shows the difference in chewing movements between an adult and child.

Murphy's Six Strokes of Mastication

Murphy (1965) studied serial cinematograph film of mastication in an Australian aboriginal subject, photographed at 64 frames per second. A recording point, the junction between mesial and incisal borders of the mandibular central incisors, was plotted frame by frame throughout four masticatory strokes. A regular pattern was outlined, although there was a wide range of variation within the pattern. The stroke can be analyzed into six phases—(1) preparatory phase; (2) contact with food bolus; (3) crushing phase; (4) tooth contact; (5) grinding phase; and (6) centric occlusion.

Preparatory phase: The ingested food is positioned by the tongue inside the oral cavity. Mandible moves toward the chewing side. Murphy identified a slight deviation to the opposite or non-food side just before the beginning of the mastication. He called this the precise beginning of the preparatory phase. This movement

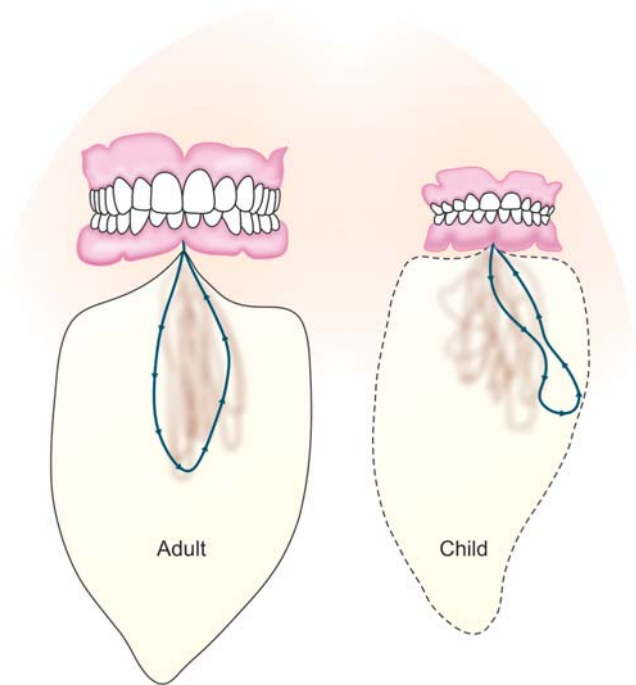


Fig. 19.5: Comparison of chewing pattern in an adult and child. In child, the jaw moves laterally first on opening while in adult, it opens vertically down followed by lateral movement

involves both a translation of the condyles due to activity of lateral pterygoid muscle which pulls the non-working condyle forward as well as a rotary or hinge movement.

Contact phase: There is a temporary hesitation in movement. This is because the sensory receptors need time to judge the consistency of the food and decide upon the force required to crush the food. Periodontal reflexes help in grasping the food in correct position between the teeth in this phase.

Crushing phase: First three or four strokes in mastication emphasize the crushing phase. This stage begins with high force and subsequently the force level declines as the food gets crushed and packed. During this phase there is equal contact on both the sides.

Tooth contact phase: Once the food particles get crushed, there is tooth contact which is accompanied by a slight change in direction of chewing. There is reduced muscular activity in masticator muscles, when this phase is reached.

Grinding phase: During this stage, the outputs from the periodontal ligament receptors reflexly control the jaw closing muscles to ensure that the teeth slide in the correct direction towards the intercuspal position. This helps to grind the food into a paste.

Centric occlusion: Centric occlusion is a connecting link between mastication and swallowing, and a position of reference to the central nervous system. It is also the beginning of the preparatory phase of the next masticatory stroke. During this phase, movement of teeth comes to a definite halt.

SPEECH

Speech unlike other orofacial functions like respiration, deglutition is a learned activity. It is a unique feature of human beings. One of the key features is the low placement of larynx in human beings which enables the human vocal tract to achieve optimal resonance to produce different sounds. The well developed velopharyngeal mechanism also aids in the speech process. Most important feature is that *Homo sapiens*' brain is prewired to develop language, when provided with a linguistic output.

Neurophysiology

The localization of language functions in the brain is a difficult task for the neurologist. Various results on sites in brain have been elucidated from subjects who acquire brain injuries with resultant loss or dysfunction in speech. It is generally believed that speech production begins in the motor area and the motor coordination of articulation is from the bilateral precentral gyri. From here nerve impulses are fed to structures in midbrain through the pyramidal tract. Here the speech is fine tuned. The cranial nerves involved in speech are trigeminal (jaw movements and craniofacial sensation), facial (circumoral muscle movement), vagus (pharyngeal and laryngeal muscles) and hypoglossal (movement of tongue). Production of speech is a highly coordinated motor task and is carried out by the interplay of numerous muscles. The language area to be discovered first was an area of the frontal lobe within the left hemisphere, called *Broca's area*, after Paul Broca (Fig. 19.6). It turns out that Broca's area is not just a matter of getting language out in a motor sense, though. It seems to be more generally involved in the ability to deal with grammar

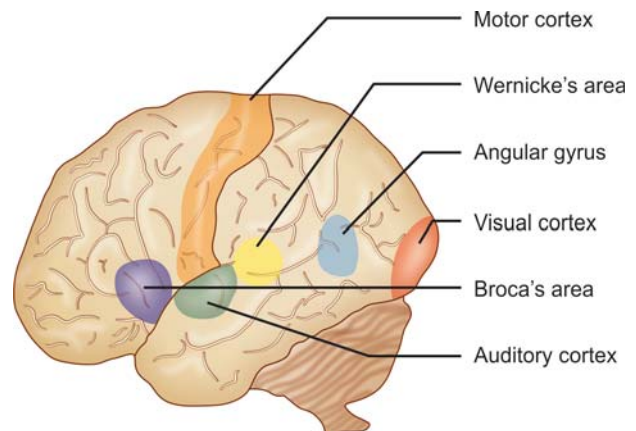


Fig. 19.6: Speech areas

itself, at least the more complex aspects of grammar. The language area to be discovered second is called *Wernicke's area*, an area at the upper portion of the temporal lobe, just behind the auditory cortex, named after Carl Wernicke, a German neurologist. Broca's and Wernicke's areas are in different lobes, yet they are actually quite near each other and intimately connected by a tract of nerves called the *arcuate fasciculus*. Physicians called the inability to speak aphasia, and the inability to produce speech was therefore called Broca's aphasia, or expressive aphasia and Wernicke's aphasia, or receptive aphasia. There is an area of the brain called the *angular gyrus* that lies about halfway between Wernicke's area and the visual cortex of the occipital lobe. The angular gyrus has been implicated in problems such as *alexia* (the inability to read), *dyslexia* (learning disability), and *agraphia* (the inability to write).

Mechanism of Speech Production

The process of producing speech sounds is as follows:

1. Lungs: fill with air.
2. Contraction of rib cage forces air from the lungs into the trachea—the volume of air determines the amplitude of the sound.
3. Trachea (windpipe): conveys air to the vocal tract. The vocal cords, at the top of the trachea, separate the trachea from the base of the vocal tract.
4. Vocal tract consists of:
 - pharynx (throat)
 - mouth
 - nose

The shape and size of the vocal tract vary by positioning the articulators: the tongue, teeth and lips. The shape of the vocal tract determines the type of speech sound, e.g. the /a/ in “hat” vs the /i/ in “hit”.

Speech differs from breathing in that at some point in the path we set the air in rapid motion or vibration during speech. Two principal components of speech production are excitation, which creates a sound by setting the air in rapid motion, and vocal tract—which “shape” the sound.

Subsystems of Speech

There are four separate functional subsystems interacting in the production of speech:

1. **Respiration:** Respiration provides the means by which larynx generates speech and voice. Airflow from lungs is essential for speech and voice production. The regular rhythm of respiration (inspiration and expiration) is affected during speech.
2. **Phonation:** This is involved in sound production. The air stream from lungs causes vibration of the vocal folds. This causes the production of raw and unmodulated sounds which gets filtered in the pharynx initially and subsequently in the oral cavity to produce proper sound.
3. **Resonance:** The sound waves produced at the vocal folds are still far from the finished product heard in speech. The resonators give the characteristic quality to the voice. The resonating structures are the air sinuses; organ surfaces; cavities such as the pharynx, oral cavity, and nasal cavity; and chest wall. The resonating structures contribute no energy to the stream of air; they act to conserve and concentrate the energy already present in the laryngeal tone rather than to let it dissipate into the tissues. Resonators shape the sounds for speech or song.
4. **Articulation:** for producing a variety of speech sounds, articulators are used. Articulation is the production of individual sounds. We manipulate and position six different articulators. They are: 1. Lips; 2. Teeth; 3. Tongue; 4. Velum (soft palate); 5. Pharyngeal wall; 6. Lower jaw.

Maturation and Description of Speech

Development of speech follows the principle of front to back maturation. The first sounds to be developed are bilabial sounds like /m/, /p/, /b/. This is the reason why

the infant’s first words are mama or papa. Figure 19.7 gives the diagrammatic representation of different sounds and Table 19.3 gives examples for different types of sounds.

Consonants and Vowels

Consonants are letters that make up most of the word whereas vowels are letters that link them together and

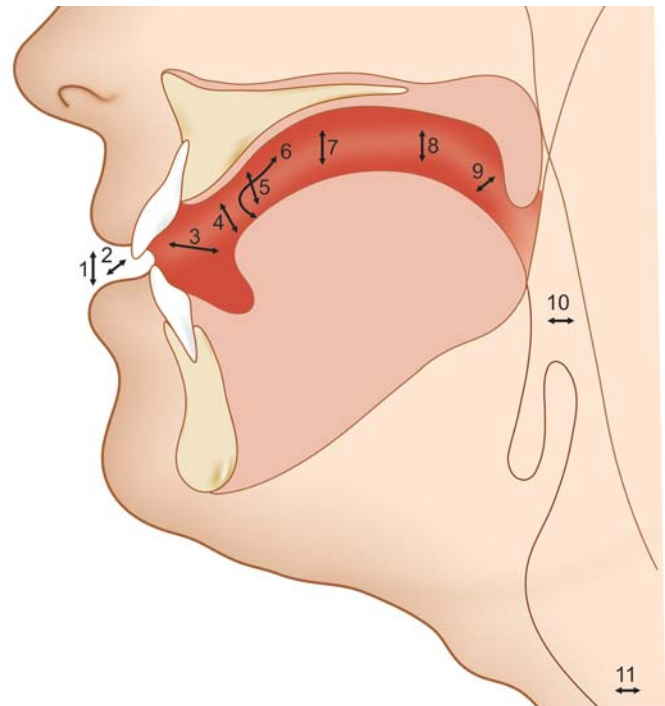


Fig. 19.7: Different types of sound: (1) Bilabial; (2) Labio-dental; (3) Linguo-dental; (4) Linguo-alveolar; (5) Linguo-palatal; (6) Retroflex; (7) Linguo-palatal; (8) Velar; (9) Uvular; (10) Pharyngeal; (11) Laryngeal

Table 19.3: Different types of speech

Type of speech	Examples
Bilabial	b, p, m
Labio-dental (upper teeth and lower lip)	f, v
Linguo-dental (upper teeth and tongue tip)	“th”, θ
Linguo-alveolar (alveolar ridge and tongue tip)	t, d, n, r
Linguo-palatal (hard palate and tongue blade)	“ch”, “sh”, “j”
Retroflex (tongue back and velum)	“ng”
Velar and uvular (tongue back, velum)	k, q, g
Pharyngeal or glottis	“h”

make them more fluent. A consonant is a speech sound that is articulated with complete or partial closure of the upper vocal tract, the upper vocal tract being defined as that part of the vocal tract that lies above the larynx. A vowel is a sound in spoken language, such as *ah*, or *oh*, pronounced with an open vocal tract so that there is no build-up of air pressure at any point above the glottis. This contrasts with consonants, such as */sh/*, where there is a constriction or closure at some point along the vocal tract.

Classes of Consonant Sounds

- **Plosives:** In this type of sound, the air stream is interrupted and suddenly released like an explosion. Examples are */p/*, */b/*, */t/* and */k/*.
- **Fricatives:** In this type of sound, the air is forced to pass through a constriction in the vocal tract. Examples are */f/*, */v/*, */“th”/* and */“sh”/* sounds.
- **Nasal:** In this type of sound, there is exit of air through the nasal cavity and not through the mouth. Examples include */m/*, */ng/*.
- **Laterals:** In this type of sound, the body of tongue elevates and air passes through right and left sides. Example is */l/*.
- **Affricates:** These sounds are combinations of plosive and fricative sounds. Example includes */ts/* as pronounced in cheese.

Milestones of Speech Development

“Milestones are a road map to the awesome processes of maturation and learning that occur in those early formative years of life”. There is a great variation in the onset of expressive language.

- Children generally understand far more (this is their “receptive speech”) than they are able to articulate themselves (“expressive language”).
- Girls seem to develop the ability to communicate earlier than boys.
- Language can develop smoothly and continuously, or in jumps and spurts.
- Because the development of speech varies, it is important not to compare a child’s language development to that of other childrens.

Milestones

- At 7 days of age, an infant can distinguish his/her mother's voice from another woman's voice.

- At 2 weeks of age, an infant can distinguish his/her father's voice from another man's voice.
- At 3 months, an infant can make vowel sounds.
- At 6 to 8 months, the infant has added a few consonant sounds to the vowel sounds, and may say “dada” or “mama,” but does not yet attach them to individuals.
- At one year, the infant will attach “mama” or “dada” to the right person. The infant can respond to one-step commands (“Give it to me”).
- At 15 months, the infant continues to string vowel and consonant sounds together (gibberish) but may imbed real words within the gibberish. The infant may be able to say as many as ten different words.
- At 18 months, a toddler can say nouns (ball, cup), names of special people, and a few action words/phrases. The infant adds gestures to his/her speech, and may be able to follow a two-step command (“Go to the bedroom and get the toy”).
- At 2 years of age, the child can combine words, forming simple sentences like “Daddy go.”
- At 3 years of age, the child can use sentences two-to four-words long, follow simple instructions, and often repeat words he/she overhears in conversations.
- At 4 years of age, the child can understand most sentences, understands physical relationships (on, in, under), uses sentences that are four- or five-words long, can say his/her name, age, and sex, and uses pronouns. Strangers can understand the child's spoken language.

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Genetics and Craniofacial Growth

CHAPTER OUTLINE

- **DNA**
 - Transcription
 - Translation
- **Gene**
- **Regulation of Gene Expression**
 - Transcription factor
- **Mutation**
- **Mendelian Genetics**
- **Modes of Inheritance**
 - Autosomal inheritance
 - Sex-linked inheritance
- **Multifactorial Inheritance**
- **Twin Studies**
 - Heritability of dentofacial phenotypes
 - Heritability of local occlusal variables
 - Heritability of tooth number, size, morphology, position and eruption
 - Limitations of twin studies
- **Methods of Transmission of Malocclusion**
- **Molecular Approach to Growth**
- **Growth Factors**
- **Trilaminar Germ Disk**
- **Neural Crest Cells**
- **The Role of Homeobox Genes**
- **Craniofacial Development**
- **Craniofacial Defects**
 - Cleft lip and cleft palate
 - Craniofacial syndromes
- **Future of Molecular Research in Craniofacial Growth**

The problems encountered in the field of genetics of growth may be divided primarily into two main categories: (1) The kinds and the relative importance of the ultimate factors, external and internal, affecting growth. (2) The modes of action of the genetic factors.

Craniofacial growth had been linked with both environmental factors and genetic factors. By the beginning of the 20th century, it became apparent that a clear understanding of the processes leading to biological evolution requires knowledge of how traits are actually transmitted from one generation to the next. Pursuit of the answer to this question led to the development of the scientific field known as genetics. Thanks to Gregor Mendel and other pioneer researchers, we now know that the mechanisms of genetic inheritance are ultimately responsible for most of the biological variation and evolutionary change. The science of genetics is concerned with the inheritance of traits, whether normal or abnormal, and with the interaction of genes and the environment. Fisher proposed the study of multifactorial inheritance in 1918. He proposed that certain traits like height are determined by a large number of segregating genes along with environmental factors. The role played by genes has been emphasized by many population studies of the twin and family methodology thereafter. Recent advances in molecular genetics have given us ample proof of the step by step molecular events resulting in multidirectional control that exists within and between the cells prenatally and post-natally, resulting in growth. The data collected so far are very little with respect to the highly complex molecular events but are still capable of generating much interest. Common structures and mechanisms of development are found among different species of animals. These include some genes, the type of genes regulation and the interaction between cells.

DNA

Deoxyribonucleic acid (DNA) contains the genetic instructions used in the development and functioning of all known living organisms. DNA is often compared to a set of blueprints, since it contains the instructions needed to construct other components of cells, such as proteins and RNA molecules. The DNA segments that carry this genetic information are called 'genes', but other DNA sequences have structural purposes, or are involved in regulating the use of this genetic information. Chemically, DNA is a long polymer of simple units called nucleotides (Fig. 20.1), with a backbone made of sugars and phosphate groups joined by ester bonds. Attached to each sugar is one of four types of molecules called bases. It is the sequence of these four bases along the backbone that encodes information. This information is read using the genetic code, which specifies the sequence of the amino acids within proteins. The code is read by copying stretches of DNA into the related nucleic acid RNA, in a process called *transcription*. Within cells, DNA is organized into structures called *chromosomes*. These chromosomes are duplicated before cells divide, in a process called *DNA replication*.

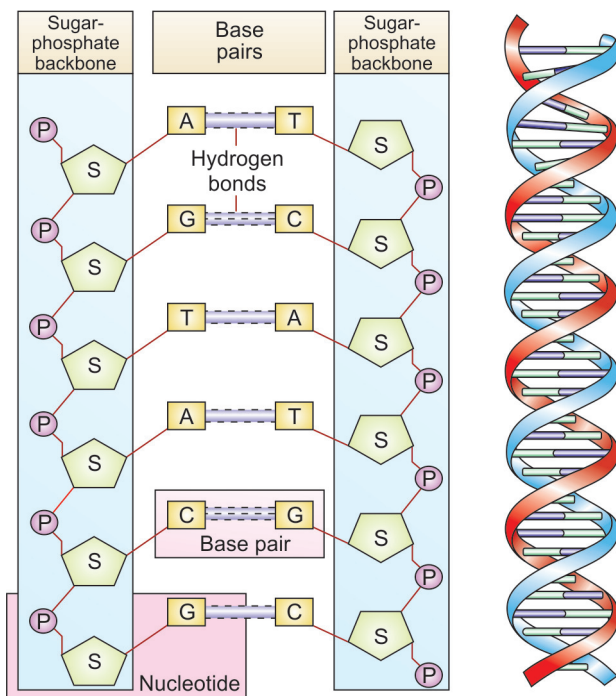


Fig. 20.1: Structure of DNA

Eukaryotes (animals, plants and fungi) store their DNA inside the cell nucleus, while in prokaryotes (bacteria) it is found in the cell's cytoplasm. Within the chromosomes, chromatin proteins such as histones compact and organize DNA. These compact structures guide the interactions between DNA and other proteins, helping control parts of the DNA that are transcribed.

The structure of DNA is a right-handed double helix. It was first described by James Watson and Francis Crick. Each spiral strand, composed of a sugar phosphate backbone and attached bases, is connected to a complementary strand by hydrogen bonding (non-covalent) between paired bases, adenine (A) with thymine (T) and guanine (G) with cytosine (C). Adenine and thymine are connected by two hydrogen bonds (non-covalent) while guanine and cytosine are connected by three hydrogen bonds.

Transcription (Fig. 20.2)

Genetic transcription results in the formation of messenger RNA (mRNA) from the DNA. The nucleotide sequence in the mRNA is complementary to the DNA. RNA polymerase is the enzyme catalyzing genetic transcription. RNA polymerase binds to the promoter area of the gene to initiate transcription. It reads the DNA template in the 3' to 5' direction and synthesizes RNA in the 5' to 3' direction. The mRNA produced by transcription is known as the *primary transcript*. It undergoes splicing of the introns (non coding regions) before being exported to the cytoplasm. This mechanism is known as the *post-transcriptional modification*. A gene can generate different transcripts which code for different proteins by a method called *alternate splicing*.

Translation (Fig. 20.2)

Translation is carried out by cell organelles, the ribosomes, which are large complexes of RNA and proteins, responsible for carrying out the chemical reactions to add new amino acids to a growing polypeptide chain by the formation of peptide bonds. They use mature mRNA molecule as a template for synthesizing new proteins. Specialized RNA molecules called transfer RNA (tRNA) is involved in translation. Each tRNA has three unpaired bases known as the *anticodon* that are complementary to the codon it reads on the mRNA. The tRNA is also attached to the amino acid

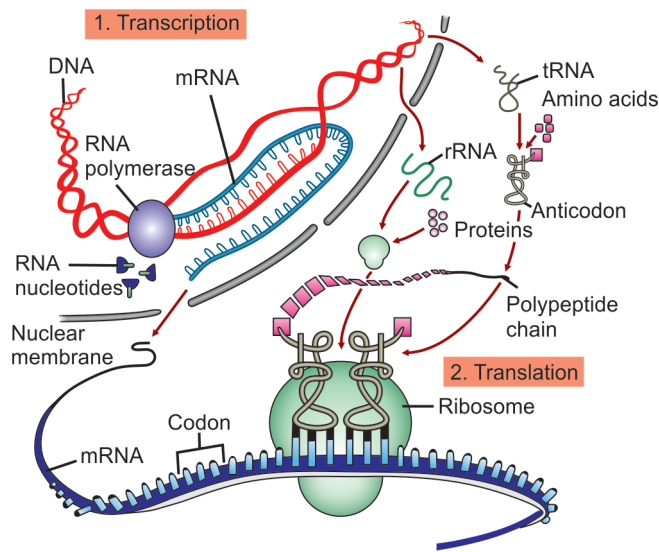


Fig. 20.2: Schematic illustration of transcription and translation resulting in protein synthesis

specified by the complementary codon. When the tRNA binds to its complementary codon in a mRNA strand, the ribosome ligates its amino acid to the new polypeptide chain, thus elongating it. The polypeptide chain is synthesized from the amino terminus to the carboxyl terminus. During and after its synthesis, the new protein achieves a three-dimensional structure to obtain functional competency.

GENE

The unit of inheritance is called a gene. According to Gerstein et al, “a gene is a union of genomic sequences encoding a coherent set of potentially overlapping functional products”. The physical development and phenotype of organisms can be thought of as a product of genes interacting with each other and with the environment. Chromosomes within the cells are the carriers of genetic material, and they are made of deoxyribonucleic acid (DNA), a polymeric molecule found in all cells.

The total set of genes in an organism is known as its genome. The estimated number of genes in the human genome has been repeatedly revised downward since the completion of the Human Genome Project. The current estimate places the human genome at just under 3 billion base pairs and about 20,000 to 25,000 genes.

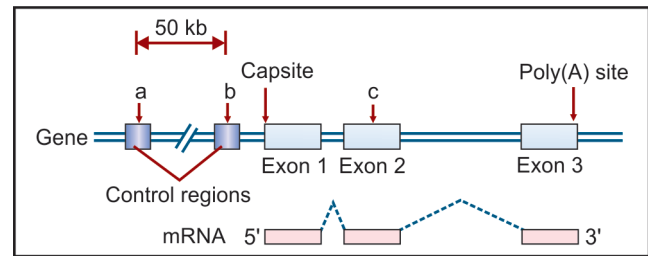


Fig. 20.3: Structure of control regions

Genes consist of a long strand of DNA which is divided into promoter, which controls the activity of a gene, and coding and non-coding sequence. The coding sequence determines what the gene produces, while the non-coding sequence is responsible for gene regulation. When a gene is active, the coding and non-coding sequence is copied in a process called transcription, producing an RNA copy of the gene's information. This RNA can then direct the synthesis of proteins by translation. Genes that encode proteins are composed of a series of three-nucleotide sequences called *codons*. Each of the codons code for an amino acid. The genetic code specifies the correspondence during protein translation between codons and amino acids. The genetic code is nearly the same for all known organisms. These molecules resulting from gene expression, whether RNA or protein, are known as gene products.

The structure of a general eukaryotic gene is a sequence of *control* regions, followed by *coding* regions (Fig. 20.3). Coding regions are sites where the promoter of inhibitor proteins might bind. The concatenation of the coding regions is the final product which goes to translation.

REGULATION OF GENE EXPRESSION

The process by which the inheritable information in a gene, such as the DNA sequence, is made into a functional gene product, such as protein or RNA is known as *gene expression*. *Gene regulation* refers to the cellular control of the amount and timing of changes in the appearance of the functional product of gene. The majority of the known mechanisms regulate the expression of protein coding genes instead of the RNA coding regions. Any step of the gene's expression may be modulated, from DNA structure to transcription to

the post-translational modification of a protein. Stages where gene expression is regulated are as follows:

- Chemical and structural modification of DNA or chromatin
- Transcription
- Translation
- Post transcriptional modification
- RNA transport
- mRNA degradation
- Post translational modifications.

Gene regulation is the basis for cellular differentiation, morphogenesis and the versatility and adaptability of any organism. It gives the cell control over its structure and function. Proteins involved in regulating gene expression are called regulatory proteins. It is usually bound to a regulatory binding site which is sometimes located near the promoter although this is not always the case. Regulatory proteins are of two types, activators or repressors. Activators bind to switch a gene on and repressors bind to shut off a gene. Generally, as the organism grows more sophisticated, their cellular protein regulation becomes more complicated and indeed, some human genes can be controlled by many activators and repressors working together.

Gene regulation can be summarized as how they respond:

Inducible systems—An inducible system is off, unless there is the presence of some molecule (called an inducer) which allows for gene expression. The molecule is said to "induce expression".

Repressible systems—A repressible system is one which except in the presence of some molecule (called a corepressor), suppresses gene expression.

Initiating the signal for gene regulation is achieved through the binding of some ligand to a receptor. Cell-surface receptors are integral transmembrane proteins and recognize the vast majority of extracellular signaling molecules. The receptors which are located both on the outside of the cell (the *extracellular domain*), and on the inside of the cell (the *intracellular domain*) spanning the plasma membrane of the cell, are called transmembrane receptors. Signal transduction occurs as a result of the stimulatory molecule or the binding of a ligand to its extracellular domain; the ligand itself does not pass through the plasma membrane prior to receptor-binding.

Transcription Factor

The initiating signal gives rise to the activation of a protein called the transcription factor. In the field of molecular biology, a transcription factor is a protein that binds to specific parts of DNA using DNA binding domains and is part of the system that controls the transfer of genetic information from DNA to RNA. Transcription factors do not need to perform this function alone, they may use other proteins in a complex, by increasing (as an activator), or preventing (as a repressor) the presence of RNA polymerase, the enzyme which activates the transcription of genetic information from DNA to RNA.

Transcription factors generally simultaneously bind DNA as well as an RNA polymerase, as well as other agents necessary for the transcription process (HATs, scaffolding proteins, etc.). Transcription factors, and their cofactors, can be regulated through reversible structural alterations such as phosphorylation or inactivated through such mechanisms as proteolysis. Transcription is initiated at the promoter site, as an increase in the amount of an active transcription factor which binds a target DNA sequence. Other proteins, known as "scaffolding proteins" bind other cofactors and hold them in place. DNA sequences far from the point of initiation, known as enhancers, can aid in the assembly of this "transcription machinery." The histone arms are acetylated, and DNA is transcribed into RNA.

Many transcription factors in multicellular organisms are involved in development. The transcription factors turn on/off the transcription of the appropriate genes which in turn allows for changes in cell morphology or activities which are needed for cell fate determination and cellular differentiation. They do this in response to the outside stimuli. For example, the transcription factor encoded by the sex determining region Y (SRY) gene which plays a major role in determining gender in humans. Another example is the HOX transcription factor family, which is important for proper body pattern formation in organisms as diverse as fruit flies to humans.

UP-REGULATION AND DOWN-REGULATION

The process which occurs within a cell, triggered by a signal originating internal or external to the cell, which results in increased expression of one or more genes and as a result, the protein(s) encoded by those genes, is called *up-regulation*. *Down-regulation* is a process

resulting in decreased gene and corresponding protein expression.

Up-regulation occurs, for example, when a cell is deficient in some kind of receptor. In this case, more receptor protein is synthesized and transported to the membrane of the cell and thus, the sensitivity of the cell is brought back to normal, re-establishing homeostasis.

Down-regulation occurs, for example, when a cell is overly stimulated by a neurotransmitter, hormone, or drug for a prolonged period of time and the expression of the receptor protein is decreased in order to protect the cell.

MUTATION

Alterations in the base sequence of a particular gene arise from a number of sources of which the more important are the errors in DNA replication and the aftermath of DNA damage. These errors are very rare. The error rate per site is only around 10^{-6} to 10^{-10} in eukaryotes. These errors are called mutations.

The cell contains many DNA repair mechanisms for preventing mutations and maintaining the integrity of the genome. Still, some situations arise such as breaks in both DNA strands of a chromosome, where repairing the physical damage to the molecule is a higher priority than producing an exact copy. Some mutations in protein-coding genes are *silent*, or produce no change in the amino acid sequence of the protein for which they code; for example, the codons UCU and UUC code for serine, so the U $\leftrightarrow$ C mutation has no effect on the protein. Mutations that do have phenotypic effects are most often neutral or deleterious to the organism. A gene's most common allele is called the wild type allele, and rare alleles are called mutants. Sometimes, mutations confer benefits to the organism's fitness. Mutations propagated to the next generation lead to variations within a species' population. Variants of a single gene are known as alleles, and differences in alleles may give rise to differences in traits. If mutation occurs during gametogenesis, the mutant allele will appear in a gamete and, consequently, in the cells throughout the body of any resulting individual. If it occurs after fertilization, as a somatic mutation, only a proportion of the cells will be affected.

Mutations of DNA (Fig. 20.4) are broadly divisible into length mutations with gain or loss of genetic material, and point mutations, with alteration of the genetic code,

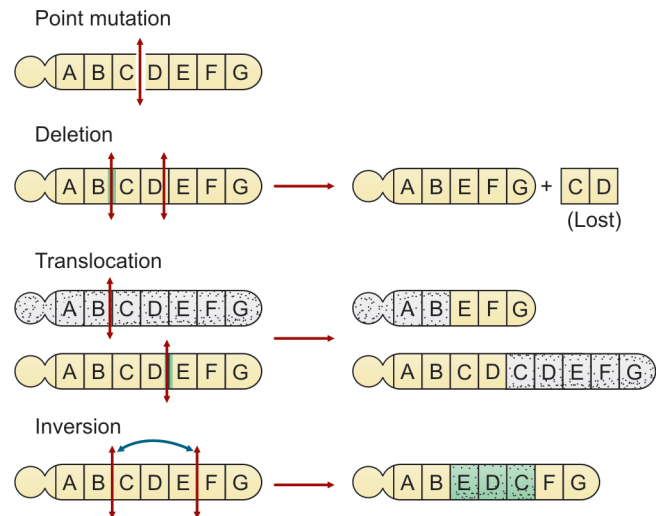


Fig. 20.4: Different types of mutation

but no gain or loss of genetic material. Large deletions remove many adjacent genes (contiguous gene disorders) and these should be suspected if a boy has several X-linked disorders or if a patient with a single-gene disorder has unexplained mental retardation and/or other congenital malformations. In a point mutation, a single nucleotide base is replaced by a different nucleotide base. Most point mutations are spontaneous and unexplained, but certain factors, such as mutagenic chemicals and ionizing radiation, can increase the spontaneous mutation rate.

Each gene is likely to influence many morphological characters so that a deleterious mutation, although producing a unitary effect at the molecular level, can result in a syndrome of morphological abnormalities. On the other hand, it is uncommon for the variants in a single gene to have clearly distinguishable phenotypic effects, since only a few traits are, in fact, controlled by single genetic loci.

Gene Targeting

Gene targeting commonly refers to the techniques used for altering or disrupting mouse genes and provides the mouse models for studying the roles of individual genes in embryonic development, human disorders, aging and diseases. The mouse models, in whom one or more of their genes were deactivated or made inoperable, are called knockout mice. Ever since the first reports in which homologous recombination in embryonic stem cells was

used to generate gene-targeted mice, gene targeting has proven to be a powerful means of precisely manipulating the mammalian genome, producing at least ten thousand mutant mouse strains and it is now possible to introduce mutations that can be activated at specific time points, or in specific cells or organs, both, during development and in the adult animal.

MENDELIAN GENETICS

The existence of genes was first suggested by Gregor Mendel. He studied inheritance in pea plants and arrived at a conclusion that traits are carried from the parent to the offspring by specific mechanisms. According to the theory of Mendelian inheritance, variations in *phenotype*—the observable physical and behavioral characteristics of an organism—are due to variations in *genotype*, or the organism's particular set of genes, each of which specifies a particular trait. Since the chromosomes exist in pairs, two copies of each gene are present. These genes may or may not show similarity to each other. Alleles are different forms of a gene, which may give rise to different phenotypes. If both copies of the gene are identical, the individual is described as *homozygous*, and if they differ, the term used is *heterozygous*. Alleles may be dominant or recessive; dominant alleles give rise to their corresponding phenotypes when paired with any other allele for the same trait. The recessive alleles give rise to their corresponding phenotype only when paired with another copy of the same allele.

Mendel's Laws

Law of Uniformity

The Law of Uniformity refers to the fact that when the homozygotes with different alleles are crossed, all the offspring in the F1 generation are identical and heterozygous.

Law of Segregation

The Law of Segregation, also known as Mendel's First Law, essentially has three parts:

1. Alternative versions of genes account for variations in inherited characteristics. This is the concept of alleles. Alleles are different versions of genes that impart the same characteristic.
2. For each characteristic, an organism inherits two alleles, one from each parent. This means that when somatic cells are produced from two alleles, one allele comes from the mother and one from the father. These alleles may be the same, or different.
3. The two alleles for each characteristic segregate during gamete production. This means that each gamete will contain only one allele for each gene. This allows the maternal and paternal alleles to be combined in the offspring, ensuring variation.

Law of Independent Assortment

The Law of Independent Assortment, also known as "Inheritance Law" or Mendel's Second Law, states that the inheritance pattern of one trait will not affect the inheritance pattern of another. Mendel, therefore concluded that different traits are inherited independently of each other, so that there is no relation between two traits in an individual.

MODES OF INHERITANCE

Autosomal Inheritance

An autosomal dominant gene is one that occurs on an autosomal (non-sex determining) chromosome. As it is dominant, the phenotype it gives will be expressed even if the gene is heterozygous. This contrasts with recessive genes, which need to be homozygous to be expressed. The chances of an autosomal dominant disorder being inherited are 50 percent if one parent is heterozygous for the mutant gene and the other is homozygous for the normal, or *wild-type*, gene. This is because the offspring will always inherit a normal gene from the parent carrying the wild-type genes, and will have a 50 percent chance of inheriting the mutant gene from the other parent. If the mutant gene is inherited, the offspring will be heterozygous for the mutant gene, and will suffer from the disorder. If the parent with the disorder is homozygous for the gene, the offspring produced from mating with an unaffected parent will always have the disorder (Fig. 20.5).

The dominant allele 'A' is passed from generation to generation. The characteristics for autosomal dominant transmission are:

- All affected individuals should have an affected parent
- Both sexes should be equally affected

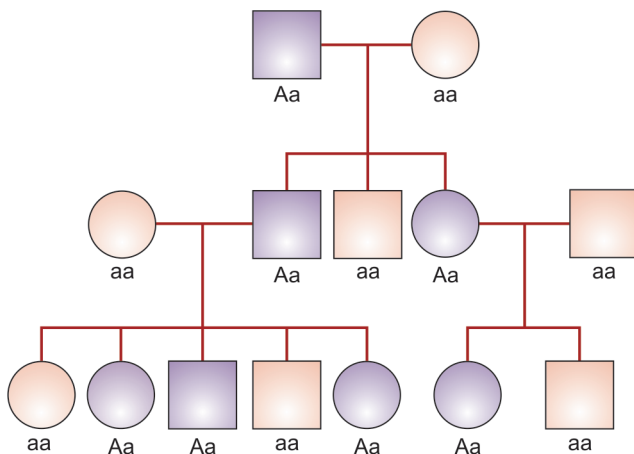


Fig. 20.5: Pedigree illustrating autosomal dominant transmission

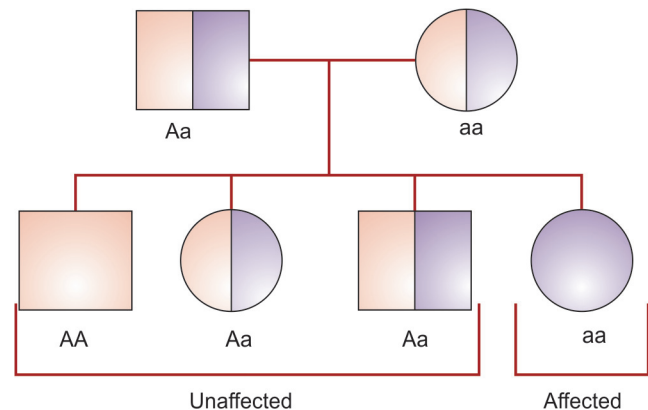


Fig. 20.6: Pedigree illustrating autosomal recessive transmission. Allele 'A' is dominant and 'a' is recessive

- Roughly 50 percent of the offspring of an affected individual should also be affected.

The term "recessive gene" refers to an allele that causes a phenotype that is only seen in homozygous genotypes and never in heterozygous genotypes. Every diploid organism, including humans, has two copies of every gene on autosomal chromosomes, one from the mother and one from the father. The dominant allele of a gene will always be expressed while the recessive allele of a gene will be expressed only if the organism has two recessive forms. Thus, if both parents are carriers of a recessive trait, there is a 25 percent chance with each child to show the recessive trait (Fig. 20.6).

The characteristics are:

- Usually, there is no previous family history.
- The most likely place to find a second affected child is a sibling of the first.
- Inbreeding increases the chance of observing an autosomal recessive condition.

Sex-linked Inheritance

Sex linkage is the phenotypic expression of an allele that is related to the sex of the individual. This mode of inheritance is in contrast to the inheritance of traits on autosomal chromosomes, where both sexes have the same probability of expressing the trait. Since, in humans, there are many more genes on the X chromosome than there are on the Y chromosome, there are many more X-linked traits than the Y-linked traits. In mammals, the female is the homogametic sex, having two

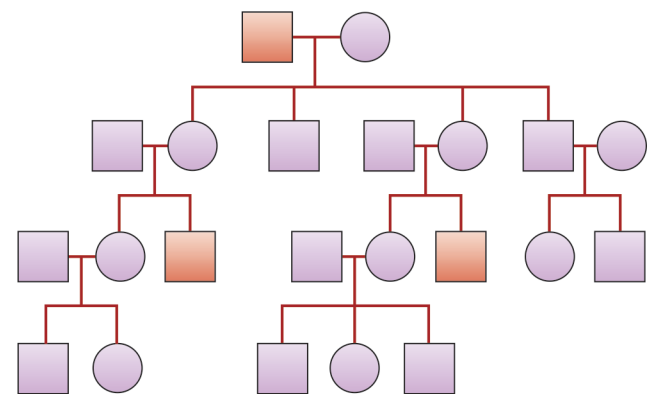


Fig. 20.7: X-linked recessive transmission

X chromosomes (XX), while the male is heterogametic, having one X and one Y chromosome (XY). Genes that are present on the X or Y chromosome are called sex linked genes.

X-linked recessive traits are expressed in all males, but only in those females which are homozygous for the recessive allele. For example, an X-linked recessive allele in humans causes hemophilia. Hemophilia is much more common in males than females because males are hemizygous—they only have one copy of the gene in question—and therefore express the trait when they inherit one mutant allele. In contrast, a female must inherit two mutant alleles, a less frequent event since the mutant allele is rare in the population (Fig. 20.7).

The X-linked recessive transmission in hemophilia. The characteristics are:

- As with any X-linked trait, the disease is never passed from father to son.
- Males are much more likely to be affected than females.
- All affected males in a family are related through their mothers.
- Trait or disease is typically passed from an affected grandfather, through his carrier daughters, to half of his grandsons.

Penetrance and expressivity are two concepts that are different yet related. They are often confused for one another. One distinguishing characteristic is that "penetrance" is a qualitative concept and "expressivity" is a quantitative concept. Simply put, *penetrance* refers to whether a phenotype is expressed for a particular genotype, and *expressivity* refers to the degree to which a phenotype is expressed when it is expressed.

MULTIFACTORIAL INHERITANCE

Many genes (many alleles) in different genetic loci are responsible for the continuous phenotypic traits, such as height, weight, or tooth size which show broad range of variability. In continuous traits, the differences are characterized quantitatively between individuals. They are further modified by environmental conditions which obscure the genetic picture. If the genetic variation of a particular phenotypic trait is dependent on the simultaneous segregation of many genes and affected by environment it is referred to as being subject to multifactorial inheritance. Each gene has a small additive effect on the expression of the trait. Also, the genes render the individual susceptible to the environmental agents. Genetic differences caused by the segregation of many genes are referred to as polygenic variation and the genes concerned are referred to as polygenes. Many congenital malformations and common diseases of adult life are inherited as multifactorial traits and these are categorized as either continuous or discontinuous.

Discontinuous Multifactorial Traits

Discontinuous multifactorial variation rests on the assumption that there is an underlying scale of continuous variation of liability to develop the condition, resulting from a combination of all the genetic and environmental

influences involved. The condition is present only when the liability exceeds a critical threshold value. An example of discontinuous multifactorial traits would be cleft lip and palate which is a congenital malformation observed in 1 in 800 births. The mildest form of the cleft lip and palate is the unilateral cleft lip. Bilateral cleft lip with complete cleft palate is the severest form of the cleft lip and palate. The parents of a cleft lip and palate are often unaffected, and there may be no family history of cleft lip and palate. The accepted explanation of the parents of cleft children is they have sufficient active genes to form cleft lip and palate. Only when the balance exceeds a certain threshold will the malformation occur, and the further the threshold is exceeded, the greater the extent of the malformation (Fig. 20.8).

Continuous Multifactorial Traits

Most of the normal human characteristics have been determined to be continuous multifactorial traits. These traits, by definition have a continuously graded distribution. Thus, for height there is a range from the very tall to the markedly short. The majority of individuals are centered around the mean. Such distribution is characteristic of a continuous multifactorial trait. Malocclusion can be considered as a continuous multifactorial trait. Malocclusion should not be regarded as abnormal or as a disease, but as a variation of occlusion in an infinite range of biological variation.

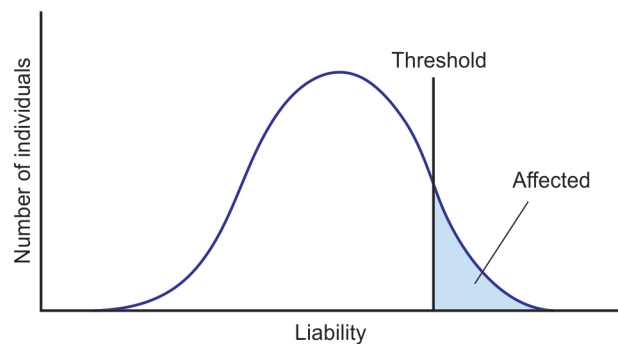


Fig. 20.8: Threshold model for multifactorial inheritance. There is a liability toward the trait that consists of a combination of genetic and nongenetic factors and is normally distributed in the population. The trait is expressed only in individuals whose liability exceeds a threshold

TWIN STUDIES

The scientific study of human twins began in the 1870s when Sir Francis Galton (1822-1911) published a series of articles arguing that heredity (nature) was a stronger factor than environment (nurture) in determining the respective characteristics of twins. Twin studies are one of a family of designs in genetics which aid the study of individual differences by highlighting the role of environmental and genetic causes on behavior. Twins are invaluable for studying these important questions because they share the genes and environments. If we observe that the children in a family are more similar than might be expected by chance, this may reflect shared environmental influences common to members of family like social class, parenting styles, education, etc. They also shared genes, inherited from parents. By studying many hundreds of families of twins, researchers can then understand more about the role of genetic effects, and the effects of shared and unique environment effects.

The power of twin designs arises from the fact that twins may be either monozygotic (MZ: developing from a single fertilized egg and therefore sharing all of their genes)—or dizygotic (DZ: developing from two fertilized eggs and therefore, sharing on an average 50 percent of their genes, the same level of genetic similarity as found in non-twin siblings). For a single-gene trait or a chromosomal disorder, the monozygotic concordance rate will be 100 percent, whereas the dizygotic rate will be less than this and equal to the rate in siblings. For discontinuous multifactorial traits with both genetic and environmental contributions, the rate in monozygotic twins, although less than 100 percent, will exceed the rate in dizygotic twins. To further simplify, observed differences within a pair of monozygotic twins (whose genotype is identical) are due to environment and those differences within a pair of dizygotic twins (who share 50 percent of their total gene complement) are due to both genotype and environment. If a condition has no genetic component, for example, due to chance or trauma, concordance rates would be expected to be similar for both types of twins. The classic twin study begins from assessing the variance of a phenotype in a large group, and attempts to estimate how much of this is due to genetic effects, how much appears to be due to shared environmental effects, and how much is

due to unique environmental effects—events occurring to one twin but not another. The twin method, when appropriately applied, provides genetists with one of the most informative techniques available for analysis of complex genetic traits. Twin studies assume that the zygosity is accurately determined and effects of the environment are equal in the two categories.

Alternative method for investigating the role of heredity in determining craniofacial morphology is by familial studies. Analysis of parent offspring correlation coefficients are used in the familial studies.

The Value of Twin Studies

Traits can be inherited through different genetic mechanisms. For traits governed by *dominant genetic* mechanisms, a dominant gene inherited from one parent triumphs a recessive gene inherited from the other parent. If a person inherits a recessive gene, for example, blue eyes from one parent and a dominant gene for brown eyes from the other parent, then the dominant brown gene wins, and the person's eyes are brown. In contrast, *additive genetic* mechanisms mix together—a plant that receives one red gene and one white gene might, if the genes are additive, turn out pink. *Epistatic* mechanisms are complex cases where interactions among multiple genes may determine the outcome of one trait. Twin studies, in general, assume that only one type of genetic mechanism—usually additive, is operating for a particular trait. Twin researchers acknowledge that these and other limitations exist. However, they say, that the limitations do not negate the usefulness of twin studies. For traits that are substantially influenced by heredity, the approximately two-fold difference in genetic similarity between the two types of twins should outweigh any complications. Twin study designs and statistical analysis methods are also constantly evolving and improving. The original twin study design has expanded to include studies of twins' extended families, longitudinal studies and other variations. Some of these variations allow researchers to address previous limitations. In the age of molecular genetics, meanwhile, the classical twin study design is only one aspect of genetics research. Twin studies estimate the heritability of a trait, but molecular genetics attempts to pinpoint the effects of a particular gene.

Heritability of Dentofacial Phenotypes

The bulk of the evidence for the heritability of various types of malocclusion arises from family and twin studies. The larger the differences between the two twin categories, the greater the genetic difference effect on variability of the trait. Polygenic inheritance implies that there is scope for environmental modification and many familial and twin studies bear this out.

Many components are involved in normal occlusion. The most important are the size of the maxilla, the size of the mandible, dental arch form, size and morphology of teeth present, and the soft tissue morphology.

Class II Malocclusion

Harris (1975) carried out cephalometric studies to determine the heritability of certain craniofacial parameters in class II division 1 malocclusion. His investigation have shown that in class II patients, the mandible is significantly more retruded than in class I patients, with the length of the body of the mandible and the overall mandibular length reduced. The study also showed a higher correlation between the patient and his immediate family than data from random pairings of unrelated siblings, thus supporting the concept of polygenic inheritance for class II division 1 malocclusion.

Class II division 2 malocclusion is a distinct clinical entity and is a more consistent collection of definable morphometric features occurring simultaneously, i.e. it is a syndrome, often accompanied by specific morphometric dental feature, such as a poorly developed cingulum on the upper incisors and a characteristic crown angulation.

Markovic (1992) carried out a cephalometric study of 114 class II division 2 malocclusions, 48 twin pairs and six sets of triplets. Of the monozygotic twin pairs, 100 percent demonstrated concordance for the class II division 2 malocclusion, whilst almost 90 percent of the dizygotic twin pairs were discordant. This is strong evidence for the fact that genetics is the main etiological factor in the development of class II division 2 malocclusion. Various studies have shown definite genetic influence in class II division 2 malocclusion, probably autosomal dominance with incomplete penetrance and variable expressivity. It could also be due to a polygenic model with simultaneous expression of number of genetically determined morphologic traits acting

additively rather than the effect of single dominant controlling gene for the entire occlusal malformation. Evidence from twin study by Lauweryns et al, 1995 has indicated strong genetic influence in masticatory muscle pattern in class II division 2 malocclusion.

Class III Malocclusion

The most significant example for hereditary transmission of class III malocclusion is the Hapsburgs jaw. Strohmayer concluded from his detailed pedigree analysis of the Hapsburg Royal family (genetic trait of large lower jaw passing through several generations in the family), that the mandibular prognathism was transmitted as an autosomal dominant trait. Various models have been suggested, since then, for the inheritance of mandibular prognathism such as autosomal dominant with incomplete penetrance, simple recessive, variable both in expressivity and penetrance with differences in different racial populations. Suzuki et al, 1961, studied 1362 persons from 243 Japanese families and noted that, while the index cases had mandibular prognathism; there was a significantly higher incidence of this trait in other members of this family (34.4%) in comparison to families of individuals with normal occlusion (7.5%). Schulze and Weise 1965, also studied mandibular prognathism in monozygotic and dizygotic twins. They reported that the concordance in monozygotic twins was six times higher than among dizygotic twins. Both of the above studies reported a polygenic hypothesis as the primary cause for mandibular prognathism

Litton et al, 1970, carried out an analysis of the literature to that date and also analyzed a group of probands, siblings and parents with class III malocclusion, and analyzed the results in an effort to determine a possible mode of transmission. Both, autosomal dominant and autosomal recessive transmission were ruled out and there was no association with gender (male or female). The polygenic multifactorial threshold model put forward by Edward et al, however, fit the data and he accordingly proposed a polygenic model with a threshold for expression to explain familial distribution, and the prevalence, both within general population and in siblings of affected persons.

Watnick, 1972, studied 35 pairs of monozygotic and 35 pairs of dizygotic like-sexed twins using lateral cephalometry. He concluded that the analysis of unit areas with the craniofacial complex represents local

growth sites and revealed different modes of control within the same bone. Certain areas, such as the lingual symphysis, lateral surface of the ramus and frontal curvature of the mandible are predominantly under genetic control. Other areas, such as the antegonial notch, are predominantly affected by environmental factors. Nakasima and Nakata, 1986, assessed the craniofacial morphologic differences between the parents of class II patients and the parents of class III patients, as well as parent-offspring correlations, and the genetic and environmental components of variation within the craniofacial complex in these malocclusions. The results showed that the parents of class II patients had a convex profile with a distocclusion type of denture pattern, while the parents of class III patients had a concave profile with a mesiocclusion type of denture pattern. This suggests that both class II and class III malocclusions have a genetic basis. The skeletal pattern was more directly related to genetic factors. Parent-offspring correlation data were in agreement with the expected level under the polygenic model of inheritance.

Upper incisor proclination, gonial angle and the ramal height were considered to be related to environmental factors.

Heritability of Local Occlusal Variables

Due to the adaptability of the dentoalveolar region when subjected to environmental factors, local malocclusions are primarily acquired and would be expected to have low heritability. Lundstrom, 1948, studied 50 pairs of monozygotic and 50 pairs of dizygotic twins and concluded that heredity played a significant role in determining, among other factors, width and length of the dental arch, crowding and spacing of the teeth and the degree of overbite. A study by Hu et al, 1992, also reported familial similarity in dental arch form and tooth position. In a recent study by King et al, 1993, initial treatment records of 104 adolescent sibling pairs, all of whom subsequently received orthodontic treatment, were examined. Heritability estimates for occlusal variations such as rotations, crossbites and displacements were significantly higher than in a comparable series of adolescents with naturally good occlusion. The explanation offered was that genetically influenced facial types and growth patterns of the siblings are likely to respond to environment factors.

Heritability of Tooth Number, Size, Morphology, Position and Eruption

Various developmental dental disorders, which are under the influence of genes, include:

1. Hypodontia
2. Supernumerary teeth
3. Abnormal tooth shape
4. Submerged primary molars
5. Ectopic eruption and Transposition of canines.

Hypodontia

The congenital absence of teeth may be referred to as hypodontia, when one or several teeth are missing, or anodontia when there is a complete absence of one or both dentitions. Features include:

- i. They are more common in permanent than primary dentition.
- ii. Absence of primary teeth associated with absence of permanent successors.
- iii. They may be associated with other developmental anomalies.

Grahnen, 1956, in his familial and twin studies, revealed the hereditary nature of hypodontia and concluded that in children with missing teeth, up to half of their siblings or parents also had missing teeth. Osborne et al, 1958, in their twin studies, have shown that the tooth crown dimensions are strongly determined by heredity. Clinical evidence suggests that congenital absence of teeth and reduction in tooth size, are associated, e.g. hypodontia and hypoplasia of maxillary lateral incisors are frequently present simultaneously. Numerous pedigrees have been published linking the two characteristics and implying that they are different expressions of the same disorder.

Gruneberg, 1965, suggested that a tooth germ must reach a critical size during a particular stage of the development or the structure will regress, and Suaraz and Spence, 1974, showed that hypodontia and reduction in tooth size are, in fact, controlled by the same or related gene loci. It is apparent from all the evidence in this respect that tooth size fits the polygenic multifactorial threshold model.

Markovic, 1982, found a high rate of concordance for hypodontia in monozygous twin pairs, while the dizygous twin pairs he observed were discordant. This, and other previous studies concluded that a single

autosomal dominant gene could explain the mode of transmission with incomplete penetrance. Dermaut and Smith, 1986, studied the prevalence of tooth agenesis correlated with jaw relationship and dental crowding in 185 patients and found that hypodontia occurred more often in girls than in boys. The upper lateral incisors and lower premolars were the most frequently missing teeth.

Class I skeletal relationships were found more often in patients with agenesis than in patients without missing teeth and are associated with deep-bite growth patterns.

As food habits are more defined with change to selective pressure during chewing there is concomitant reduction in tooth volume in the respective fields of incisors, premolars and molars. Therefore, hypodontia involving third molars, second premolars and lateral incisors are more common. This has been referred to as "*Butler's field theory*".

Supernumerary Teeth

These are teeth additional to those of the normal complement of teeth. A mesiodens is a supernumerary tooth occurring between the maxillary central incisors and is the most common of all supernumerary teeth. Supernumerary teeth most frequently seen in the pre-maxillary region and with a male sex predilection also appear to be genetically determined. Niswander and Sugaku, 1963, analyzed the data from family studies and have suggested that, like hypodontia, the genetics of the less prevalent condition of supernumerary teeth is under the control of number of different loci. Brook, 1980, found that mesiodens is more commonly present in parents and siblings of patients who present with mesiodens, although inheritance does not follow a simple Mendelian pattern. Evidence from twins with supernumerary teeth also supports this theory (Jasmin et al, 1993).

Abnormal Tooth Shape

Alvesalo and Portin, 1992, provided substantial evidence supporting the view that missing and malformed lateral incisors may be the result of a common gene defect. Abnormalities in the lateral incisor region varies from peg shaped to microdont to missing teeth, all of which have familial trends with female preponderance, and association with other dental anomalies, such as other missing teeth, ectopic canine, and transposition,

suggesting a polygenic etiology. Aspects of tooth morphology such as the Carabelli trait also seem to be strongly influenced by genes as evidenced by Australian twin study by Townsend and Martin, 1992.

Submerged Primary Molars

Primary molar submergence occurs most often in the mandibular arch with a wide variation in the reported population. Helpin and Duncan, 1986 found that, the siblings of children with submerged primary molars are also likely to be affected and in monozygous twins there is a high rate of concordance indicating a significant genetic component in the etiology. It is also of interest that a variety of abnormalities are also associated with tooth submergence, with a suggestion that this may encompass different manifestations of one syndrome, each manifestation having incomplete penetrance and variable expressivity.

Ectopic Eruption and Transposition of Canines

Various studies in the past have indicated a genetic tendency for ectopic maxillary canines. Zilberman et al, 1990, concluded that palatally placed ectopic canines are an inherited trait, being one of the anomalies in a complex of genetically related dental disturbances often occurring with missing teeth, tooth size reduction, and other ectopically positioned teeth. Previous studies by Mossey et al, 1994, have also shown an association between ectopic-maxillary canine and class II division 2 malocclusion, a genetically inherited trait. Peck et al, 1997, classified a number of different types of tooth transposition in both maxillary and mandibular arches, with maxillary canine/first premolar transposition being the most common. They also provided strong evidence of a significant genetic component as the cause of this most common type of transposition, in that, there was a familial occurrence, bilateral occurrence in a high percentage of cases, female predominance and a difference in different ethnic groups. An increased frequency of associated dental anomalies; tooth agenesis and peg-shaped maxillary lateral incisors were also reported.

Cleft Lip and Palate

Connor and Ferguson-Smith, 1993, studied cleft lip and palate and isolated cleft palate. The monozygotic twin

concordance rate for CL(P) and for CP is 35 and 26 percent, respectively, and for dizygotic twins 5 and 6 percent, respectively. This reflects the heritability of the condition: The higher the monozygotic concordance, the more important the genetic contribution, and so the higher the heritability.

LIMITATIONS OF TWIN STUDIES

The Twin Method has been subject to criticism from statistical genetics, statistics and psychology, with some arguing that the conclusion reached via this method is ambiguous or meaningless. The core elements of these criticisms are listed below:

1. It has been argued that the statistical underpinnings of twin research are invalid. Such statistical critiques argue that heritability estimates used for most twin studies rest on restrictive assumptions which are usually not tested, and if they are, are often found to be violated by the data.
2. The results of twin studies cannot be automatically generalized beyond the population in which they have been derived. It is, therefore, important to understand the particular sample studied, and the nature of twins themselves.
3. For very obvious reasons, studies of twins are with almost no exceptions, observational. This contrasts with, for instance, studies in plants or in animal breeding where the effects of experimentally randomized genotypes and environment combinations are measured. In human studies, we observe, rather than control, the exposure of individuals to different environments.

Rosario HY Potter, has established the following three guidelines for twin studies:

1. Mean trait size should not be associated with zygosity; average trait size should be statistically equivalent between samples of MZ and DZ twins. Otherwise, all subsequent analytic steps—including estimating heredity can be biased. Moreover, if the variances differ, the research needs to focus on why mean size is modulated by zygosity.
2. The total variance between zygosity must be equal. Again, significant association between variance and zygosity will influence results and it cannot be determined without analysis whether the difference will bias the estimates upward or downward. Obviously, ignoring the problem by not testing for

these common biases does not make them go away, it just makes the end result unreliable.

3. The association (covariance) between environment and zygosity must be equal in the MZ and DZ samples. That is, an important assumption of the twin model is that sharing the same prenatal and postnatal environments should not cause children within MZ twin pairs to be more similar than children within DZ pairs. Indeed, there are numerous environmental influences that should cause the phenotypes of MZ twins to converge relative to DZ pairs.

METHODS OF TRANSMISSION OF MALOCCLUSION

Malocclusions are transmitted by three different ways from genetic point of view. They are:

- Repetitive
- Discontinuous
- Variable.

Repetitive: Recurrence of a single dentofacial deviation within the immediate family and in the progenitors.

Discontinuous: Recurrence of a tendency for a malocclusal trait to reappear after few generations. Some generations will be skipped and the tendency later reappears within family.

Variable: Occurrence of different, but related, types of malocclusion within the several generations of the same family.

MOLECULAR APPROACH TO GROWTH

Molecular genetics means the reinterpretation of Mendelian genetics in molecular terms. Molecular genetics is the study of the agents that pass information from generation to generation. These molecules, genes, are long polymers of deoxyribonucleic acid.

GROWTH FACTORS

The term growth factors denominates a group of polypeptides which are involved in cellular proliferation, differentiation and morphogenesis of tissues and organs during embryogenesis, postnatal growth and adulthood. Growth factors are proteins that bind to the receptors on the cell surface, with the primary result of activating cellular proliferation and/or differentiation. Many growth factors are quite versatile, stimulating cellular division in

numerous different cell types; while others are specific to a particular cell-type. Growth factors comprise molecules that function not only as growth stimulators but also as growth inhibitors, factors that stimulate cell migration or as chemotactic agents or inhibit cell migration or invasion of tumor cells, factors that modulate differentiated functions of cells, factors involved in apoptosis, factors involved in angiogenesis, or factors that promote survival of cells without influencing growth and differentiation.

The effect of a growth factor is mediated through surface receptors on the target cells by activating intracellular phosphorylating enzymes, which in turn, induce an intracellular signaling pathway by aggregation of co-factors and other proteins which migrate to the nucleus. Together with other transcription factors they activate a set of genes, which then exert the specific changes in cellular activity or phenotype. *In vivo*, the effect of growth factors is regulated through a complex system of feedback loops, which involve other growth factors, enzymes and binding proteins.

Transforming Growth Factor (TGF)

Transforming growth factor is used to describe two classes of polypeptide growth factors, TGF α and TGF β . The name "Transforming Growth Factor" is somewhat arbitrary, since the two classes of TGFs are not structurally or genetically related to one another, and they act through different receptor mechanism.

TGF α is upregulated in some human cancers. It is produced in macrophages, brain cells, and keratinocytes, and induces epithelial development. TGF β exists in three known subtypes in humans, TGF β 1, TGF β 2, and TGF β 3. These are upregulated in some human cancers, and play crucial roles in tissue regeneration, cell differentiation, embryonic development, and regulation of the immune system. Isoforms of transforming growth factor-beta (TGF- β 1) are also thought to be involved in the pathogenesis of pre-eclampsia, TGF β receptors are single pass serine/threonine kinase receptors.

The TGF- β family is part of a super family of proteins known as the transforming growth factor beta superfamily, which includes inhibins, activin, anti-müllerian hormone, bone morphogenetic protein, etc. TGF beta controls proliferation, cellular differentiation, and other functions in most cell types.

Fibroblast Growth Factors (FGF)

Fibroblast growth factors (FGF) are a family of growth factors involved in angiogenesis, wound healing, and embryonic development. The FGFs are heparin-binding proteins and interactions with cell-surface associated heparin sulfate proteoglycans have been shown to be essential for FGF signal transduction. FGFs are key-players in the processes of proliferation and differentiation of cells, particularly endothelial cells; they (especially FGF-1) promote angiogenesis. In humans, 23 members of the FGF family have been identified, all of which are structurally related signaling molecules. The fibroblast growth factor receptor family consists of 4 members, FGFR1, FGFR2, FGFR3, and FGFR4. One of the most important functions of aFGF (FGF-1) and bFGF (FGF-2) is the promotion of endothelial cell proliferation and the physical organization of endothelial cells into tube-like structures. It thus, promotes angiogenesis, the growth of new blood vessels from the pre-existing vasculature. aFGF is a more potent angiogenic factor than VEGF (vascular endothelial growth factor) or PDGF (platelet-derived growth factor). As well as stimulating blood vessel growth, aFGF and bFGF are important players in wound healing. They stimulate angiogenesis and the proliferation of fibroblasts that give rise to granulation tissue, which fills up a wound space/cavity early in the wound healing process.

It has also been demonstrated that fibroblast growth factors are associated with many developmental processes including mesoderm induction, antero-posterior patterning, neural induction, angiogenesis, axon extension and limb formation. FGFs are crucial for the normal development of both vertebrates and invertebrates and any irregularities in their function leads to a range of developmental defects.

Associated Syndromes (Table 20.1)

Craniosynostosis syndromes have been shown to result from mutations in FGFR1, FGFR2 and FGFR3. Sometimes, the same mutation can cause two or more different craniosynostosis syndromes.

Insulin-like Growth Factors (IGFs)

The insulin-like growth factors (IGFs) are polypeptides with high sequence similarity to insulin. IGFs are part of a complex system that cells use to communicate with

Table 20.1: Syndromes and affected growth factors

<i>Affected receptor</i>	<i>Syndromes</i>	<i>Phenotypes</i>
FGFR1	Pfeiffer	Broad first digits, hypertelorism
FGFR2	Apert	Mid-face hypoplasia, fusion of digits
FGFR2	Beare-Stevenson	Mid-face hypoplasia, corrugated skin
FGFR2	Crouzon	Mid-face hypoplasia
FGFR2	Jackson-Weiss	Mid-face hypoplasia, foot anomalies
FGFR2	Pfeiffer	Same as for FGFR1 mutations
FGFR3	Crouzon	Mid-face hypoplasia, acanthosis nigricans
FGFR3	Non-syndromic craniosynostosis	Digit defects, hearing loss.

their physiologic environment (IGF axis). This complex system consists of two cell-surface receptors (IGF1R and IGF2R), two ligands (IGF-1 and IGF-2), a family of six high-affinity IGF binding proteins (IGFBP 1-6), as well as associated IGFBP degrading enzymes, referred to collectively as proteases.

The IGFs are known to bind the IGF-1 receptor, the insulin receptor, the IGF-2 receptor, the insulin-related receptor and possible other receptors. The IGF-1 receptor seems to be the "physiologic" receptor—IGF-1 binds to it with significantly higher affinity than which it binds with to the insulin receptor. Like the insulin receptor, the IGF-1 receptor is a receptor tyrosine kinase—meaning, the receptor signals by causing the addition of a phosphate molecule on particular tyrosines.

IGF-1 and IGF-2 are regulated by a family of proteins known as the IGF binding proteins. These proteins help to modulate IGF action in complex ways that involve both inhibiting IGF action by preventing binding to the IGF-1 receptor, as well as promoting the IGF action possibly through aiding in delivery to the receptor and increasing IGF half-life.

IGF-1/Growth Hormone Axis

Insulin-like growth factor 1 (IGF-1) is mainly secreted by the liver as a result of stimulation by growth hormone (GH). IGF-1 is important for both the regulation of normal physiology, as well as a number of pathological states, including cancer. The IGF axis has been shown to play roles in the promotion of cell proliferation and the inhibition of cell death (apoptosis). Gene knockout studies in mice have confirmed this, though other animals are likely to regulate the expression of these genes in distinct ways. Factors that are known to cause variation in the levels of GH and IGF-1 in the circulation include an individual's genetic make-up, the time of day, their age, sex, exercise status, stress levels, genetics, nutrition

level and body mass index (BMI), disease state, race, estrogen status, etc. Insulin-like growth factor 2 (IGF-2) is thought to be a primary growth factor required for early development while IGF-1 expression is required for achieving maximal growth. While IGF-2 may be primarily fetal in action, it is also essential for the development and function of organs such as the brain, liver and kidney.

Bone Morphogenetic Proteins (BMPs)

Bone Morphogenetic Proteins (BMPs) are a group of growth factors and cytokines known for their ability to induce the formation of bone and cartilage. Originally, seven such proteins were discovered. Of these, six of them (BMP2 through BMP7) belong to the Transforming growth factor beta superfamily of proteins. Since then, thirteen more BMPs have been discovered, bringing the total to twenty.

BMPs interact with specific receptors on the cell surface, referred to as bone morphogenetic protein receptors (BMPRs). They have an important role during embryonic development on the embryonic patterning and early skeletal formation. As such, disruption of BMP signaling can affect the body plan of the developing embryo. For example, BMP4 and its inhibitors *noggin* and *chordin* help in regulating the polarity of the embryo. Mutations in BMPs and their inhibitors (such as sclerostin) are associated with a number of human disorders which affect the skeleton. Several BMPs are also named 'cartilage-derived morphogenetic proteins' (CDMPs), while others are referred to as 'growth differentiation factors' (GDFs).

TRILAMINAR GERM DISK

Development begins with fertilization, the process by which male gamete, the sperm, and the female gamete, the oocyte, unite to give rise to the zygote. Establishment

of body axes take place during the third week of development (trilaminar germ disc stage). The three germ layers in the trilaminar disk, namely ectoderm, endoderm and mesoderm are formed by a process called gastrulation.

1. The cells at the anterior margin of the embryonic disk are called the anterior visceral endoderm. The cells at this region express genes OTX2, L1M1, and HESX1 and they signal the anteroposterior axis even before gastrulation.
2. Gastrulation begins with the formation of primitive streak. Primitive streak is initiated and maintained by nodal which is a member of TGF- β family.
3. BMP-4 is secreted throughout the embryo at this stage. Ventralisation of the mesoderm to form kidneys, blood and body wall take place under the influence of BMP-4 in the presence of FGF.
4. Chordin, noggin and follistatin are expressed in the primitive node. They oppose the activity of BMP-4 and dorsalise the cranial mesoderm to form notochord, somites and somitomeres. Inhibition of BMP-4 is facilitated by Goosecoid. Inhibition of BMP-4 by the aforementioned mechanism also leads to neural induction.
5. FGF-8 expresses nodal in the left side of the embryo and is stimulator for the left and right sidedness. Nodal upregulates PITX2 which is responsible for the left sidedness.

NEURAL CREST CELLS

The neural crest, a transient component of the ectoderm, is located inbetween the neural tube and the epidermis (or the free margins of the neural folds) of an embryo during neural tube formation. Neural crest cells quickly migrate during, or shortly after neurulation, an embryological event marked by neural tube closure.

Blockage of the activity of BMP-4 has been shown to dorsalise the cranial mesoderm to form the notochord. Appearance of notochord causes the overlying ectoderm to thicken to form neural plate. By the end of third week, the lateral edges of the neural plate thicken to form neural folds and the depressed midregion forms the neural groove. The neural folds elevate and fuse, forming the neural tube. The cells at the lateral border of the neural folds called neural crest cells dissociate from the neuroectoderm and enter the underlying mesoderm by active migration and displacement. The neural crest cells undergo epithelial to mesenchyme transition as it leaves the neuroectoderm.

The neural crest is a highly pluripotent cell population which plays a critical role in the development of the vertebrate head. Unlike most parts of the body, the facial mesenchyme is derived principally from the neural crest and not the mesoderm of the embryonic third germ layer. Neural crest cells migrates extensively throughout the embryo in four overlapping domains (cephalic, trunk, sacral and cardiac) and in the developing head the cephalic neural crest migrates from the segmented hindbrain regions (rhombomeres) into the branchial arch system. There are eight of these segments in the hindbrain (R1 to R8). The ectomesenchymal neural crest cells interact with epithelial and mesodermal population present within the arches, leading to the formation of craniofacial bone, cartilage and connective tissue.

The neural crest cells from specific segments populate specific pharyngeal arches (Fig. 20.9). Crest cells from

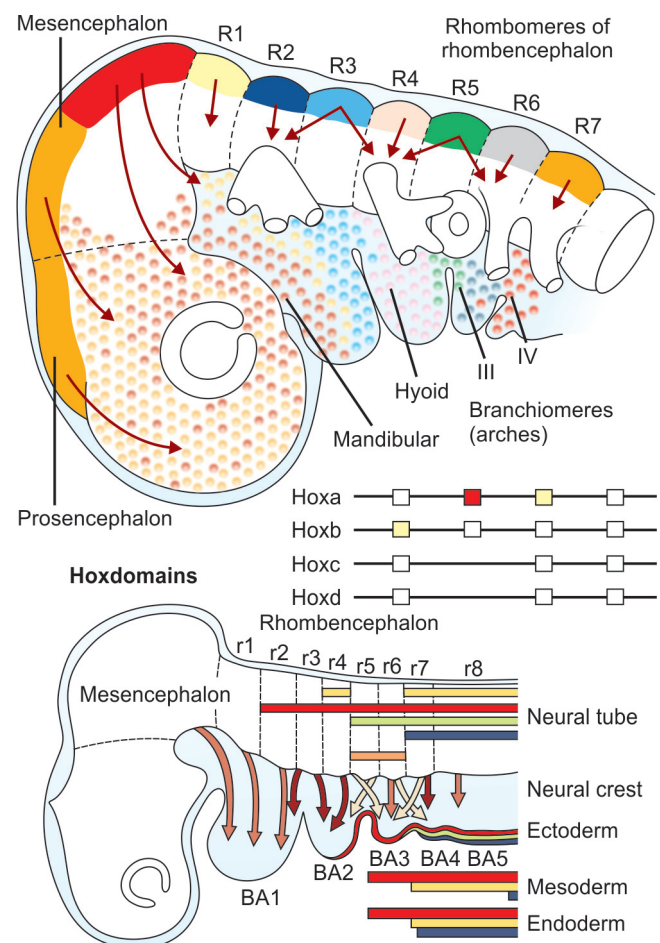


Fig. 20.9: Patterns of neural crest cell migrations into pharyngeal arches and Hox gene expression

R1 and R2 migrate to first arch, R4 to the second, R6 and R7 to third, R8 to fourth and sixth arches. The first arch also receives crest cells from midbrain.

THE ROLE OF HOMEBOX GENES (FIGS 20.10A AND B)

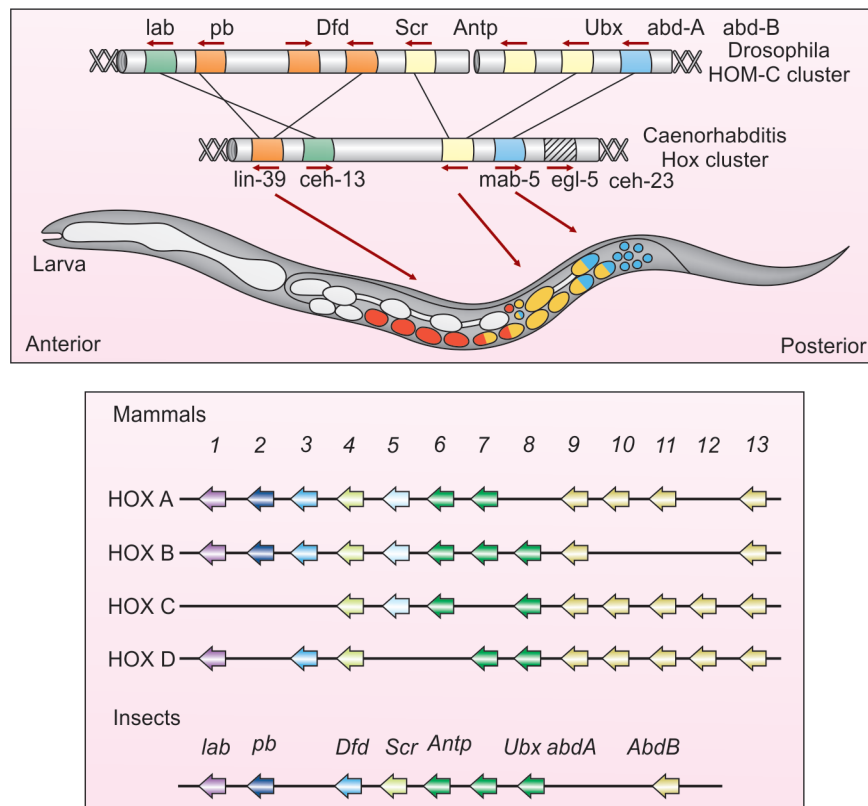
A *homeobox* is a DNA sequence found within genes which are involved in the regulation of development (morphogenesis) of animals, fungi and plants. Genes that have a homeobox are called *homeobox genes* and form the *homeobox gene family*. Homeobox genes are highly conserved throughout the evolution of diverse organisms and are now known to play a role in patterning the embryonic development. These can also be regarded as the master genes of the head and face controlling patterning, induction, programmed cell death, and epithelial mesenchymal interaction during the development of the craniofacial complex. The differences

between different organisms can be explained by the different modes of action of the homeobox genes.

Those of particular interest in the craniofacial development include the HOX group, MSX1 and MSX2 (muscle segment), DLX (distalless), OTX (orthodontical), GSC (goosecoid), and SHH (sonic hedgehog). There are four HOX gene copies in humans, existing as HOXA, HOXB, HOXC and HOXD. Each copy lies on a separate chromosome and the genes in each copy can be numbered from 1 to 13. Homeobox genes have the following properties:

- Encode transcription factors.
- Each has a DNA-binding homeodomain.
- Act in sequential zones of the embryo in the same order that they occur on the chromosome.

Homeobox genes encode proteins named transcription factors which control the transcription of RNA from the DNA template within the cell nucleus.



Figs 20.10A and B: Arrangement of homeobox genes of (HOM-C) classes of *Drosophila melanogaster* (fruit fly), HOX cluster of *Caenorhabditis* and the conserved homologous genes in the same class of humans. The genes have been duplicated during evolution such that humans have four copies on different chromosomes. The genes with the same number but positioned on the same chromosome form a paralogous group

Transcription factors can turn genes on and off by activating or repressing gene expression. They, therefore, control other genes producing a co-ordinated cascade of molecular events which, in turn, control patterning and morphogenesis. At the cellular level this control is expressed through two main groups of regulatory proteins, the growth factor family steroid/thyroid/retinoic acid super family. These regulatory molecules in the mesenchyme such as fibroblast growth factor (FGF), epidermal growth factor (EGF), transforming growth factor alpha (TGF α), transforming growth factor beta (TGF β), and bone morphogenetic proteins (BMPs) are the vehicles through which the homeobox gene information is expressed in the co-ordination of cell migration and subsequent cell interactions that regulate growth. From this, it can be inferred that the different parts of the DNA are activated in different cells, regulating the different proteins, enzymes, etc. produced by different tissues and organs.

The expression of the HOX genes is evident in the hindbrain, but it is also evident in the neural crest cells which populate the branchial arches, providing further evidence that hindbrain segmentation has direct effects on craniofacial patterning. Patterning of the pharyngeal arches, except the first arch, is regulated by the HOX genes carried in the neural crest cells. HOX gene expression in the hind brain takes place in overlapping patterns. These expression patterns determine organization of cranial ganglia and nerves. Crest cells express HOX gene from the segment of origin but the maintenance of this expression is dependent upon the interaction with the cells of mesoderm in the respective pharyngeal arches. *Sonic hedgehog* (SHH) has been shown to regulate HOX gene expression in the arches, as is the case with *retinoic acid*. Retinoic acid regulates HOX genes through retinoic acid response elements (RARE) in a concentration dependent manner.

CRANIOFACIAL DEVELOPMENT

Patterning of Face and Jaws

In humans a number of homeobox-containing genes are expressed in the maxillary, mandibular arches, and the developing facial primordia. These genes include MSX-1, MSX-2, DLX1-6 and BARX-1. Again, many of these homeobox-containing genes are related to the families of genes found in *Drosophila melanogaster*.

Knockout studies in mice have confirmed that these genes perform essential roles during the formation of the facial complex.

Members of the MSX gene family (MSX-1 and MSX-2) are normally expressed strongly in the neural crest derived mesenchyme of the developing facial prominence, and there is now strong evidence of the role of these genes in specification of the skull and face. Targeted disruption of MSX-1 in the mouse produces a number of defects in facial structures. There is cleft palate associated with a loss of the palatine bones, maxillary and mandibular hypoplasia, and a highly penetrant arrest of tooth formation at the bud stage of tooth development.

In mice, the defects in MSX-2 cause skull ossification with the persistence of calvarial foramen. This arises as a result of defective osteoprogenitor proliferation during calvarial morphogenesis. The osteocalcin gene, expressed uniquely in osteoblastic cells, is dependent on the binding of MSX gene products. Expression of MSX2 is studied in the osteoblasts of the periosteum of the mature mandible and maxilla. Expression of MSX2 is a clear marker differentiating the mature mandibular and maxillary alveolar bones from other bones of the adult skeleton.

The MSX gene is involved in tooth formation (epithelial mesenchymal tissue interactions). MSX1 is expressed in migrating neural crest cells and in mesenchymal cells of dental papilla and follicles. Three MSX genes in mammals are MSX1-MSX3. MSX1 knockout in mice caused—clefting, aberration in tooth development, missing teeth and deficiency of alveolar bones. MSX2 knockout—multiple inductive failures and early death. The combination of MSX1 and 2 knockout—causes severe aberration of development of the skeleton and some other organs, severe skeletal deficiencies in calvaria, teeth and alveolar bone.

Members of the multi-gene DLX family are expressed in a complex pattern within the embryonic ectoderm and mesenchyme of the maxillary and mandibular processes of the first arch. Targeted mutation in DLX-1, DLX-2 and DLX 1/2 provide evidence that these genes are required for the development of neural crest derived skeletal elements of the first and second branchial arches. Analysis of these mutations reveals that DLX-1 and DLX-2 regulate proximal first arch structures and that, in the mandibular primordium, there is considerable functional

redundancy of DLX-1 and DLX-2 with other members of the DLX family.

Gooseoid is another homeobox-containing transcription factor known to be ultimately responsible for the organization of the complete body axis in the early embryo. However, when gooseoid was knocked out in transgenic mice they formed a body axis normally, but exhibited a number of craniofacial defects. In wild type mice, gooseoid transcripts had been detected at the later stages of development in the osteogenic mesenchyme of the developing mandible, tongue and middle ear. In mutants, the mandible was hypoplastic, and lacked coronoid and angular process, whilst there were defects in several bones, including the maxillary, the palatine, and the pterygoid. As a homeobox-containing transcription factor, it would appear that gooseoid is involved in essential inductive tissue interactions during the formation of the head.

Endothelin, another gene that has produced an even more perplexing phenotype Endothelin-1 which encodes a vasoactive peptide expressed in vascular endothelial cells and is thought to play a role in the regulation of blood pressure. Mice with targeted disruption of endothelin-1 have no abnormalities of their cardiovascular system, but do have a marked reduction in tongue size, micrognathia and cleft palate. One of the two G protein-coupled endothelin receptors, ET-A is expressed in the neural crest derived ectomesenchyme of the branchial arches, whilst its primary ligand, ET-1 is expressed in arch epithelium, pharyngeal pouch endothelium, and arch core paraxial mesoderm. The ET-A/ET-1 pathway appears to be important for the proper patterning of the caudal regions of the first arch. It has been recently shown that the craniofacial defects in ET-A mice are, in part, due to the absence of the gooseoid transcription factor.

Patterning the Midline

Sonic hedgehog (SHH) is the vertebrate homologue of the *Drosophila* hedgehog segment polarity gene. Hedgehog morphogenes are involved in the control of left-right asymmetry, the determination of polarity in the central nervous system, somites and limbs, and in both organogenesis and the formation of the skeleton. In the vertebrate embryo, SHH encodes a signaling peptide which is involved in a number of well-characterized developmental signaling centers.

Recently, clues about the regulation of craniofacial morphogenesis have come from studies of SHH gene. Mutations of SHH in the mouse and human leads to profound abnormalities in craniofacial morphogenesis. Loss of SHH produces defective patterning of the neural plate resulting in holoprosencephaly, a failure of cleavage in the midline of forebrain and cyclopia. Later in development, SHH is expressed in the ectoderm of the fronto-nasal and maxillary processes and has been shown to be essential for their normal development. By manipulating developing chick embryos, it has been shown that a transient loss of SHH signaling in these regions of the developing face can result in defects analogous to hypotelorism and cleft lip/palate, which are characteristic features of the milder form of holoprosencephaly. In contrast, excess SHH leads to mediolateral widening of the frontonasal process resulting in hypertelorism. In severe cases, this can lead to facial duplication.

Growth of Craniofacial Skeleton

The neurocranium, which includes both the cranial vault (desmocranium) and the cranial base (chondrocranium), is characterized by rapid and significant expansion early in postnatal life. Growth of the neurocranium is characterized by skeletogenesis at the cranial base synchondroses and at the cranial vault sutures. Growth of the midfacial skeleton occurs mainly at the facial sutures. Skeletal remodeling accounts for additional size and shape change of the cranial vault and facial skeleton after the end of the second decade in humans, and may actually continue to a small degree throughout life, especially in males. Due to the inherent differences between cartilage and bone, growth and transcription factor regulation of cranial base expansion through synchondral growth is expected to be different from their regulation of membranous bone growth at the sutures. However, the growth and transcription factor regulation of bone growth at the cranial and facial sutures would be expected to be similar.

Growth of Cranial Base

The postnatal growth of the cranial base occurs by expansion at the cranial base synchondroses, principally through the intrinsic growth potential of the cartilaginous synchondroses, rather than as a secondary response to

outside stimuli. Growth is initiated and proceeds by continuous growth factor signaling between the cells of the perichondrium and the chondrocytes of the cartilages. Once expansion occurs within the cartilage, the cartilage is replaced by bone via endochondral bone formation, similar to that seen in the growth of epiphyseal plates in long bones. In contrast, postnatal growth of the cranial and facial membranous bones occurs secondarily in response to extrinsic factors. These extrinsic factors induce bone formation at the edges of the bones on either side of the fibrous sutures uniting the membranous bones. It is generally thought that bone growth at the cranial and facial sutures occurs when the bone fronts are gradually forced apart by the expansion of the underlying tissues such as the brain, dura mater, and nasal cartilages, thus exerting an overall tensile force at the sutural growth sites. However, even this has an underlying intrinsic or genetic component. In the cranial vault, the primary growth stimulus is the expanding brain. As the brain expands and the cranial base synchondroses lengthen, the cranial sutures respond by adding intramembranous bone at the edges of the bone fronts. There are, therefore, developmental relationships between the cranial base growth centers and the other craniofacial skeletal and soft tissue structures, especially the cranial sutures. As a result, defects in the growth and development of the cranial sutures may be reflected as secondary malformations in the cranial base. On the other hand, primary malformations of the cranial base synchondroses may cause craniosynostoses secondarily. These secondary deformations hypothetically would result from the transmission of aberrant mechano-tensile forces through the dura mater leading to constricted cranial sutures.

The broad mechanisms so far described in cartilage growth at the cranial base are as follows:

1. Parathyroid-hormone-related peptide (PTHrP) stimulates chondrocyte proliferation and inhibits chondrocyte hypertrophy.
2. Indian hedgehog (IHH) controls both chondrocyte proliferation and hypertrophy through molecular circuitry with PTHrP and parathyroid hormone receptor (PTHR).
3. Bone morphogenetic proteins (BMPs) stimulate chondrocyte differentiation, hypertrophy, and mineralization.
4. Fibroblast growth factors (FGF), signaling through fibroblast growth factor receptors (FGFR), inhibit chondrocyte proliferation.
5. Transforming growth factor beta (TGF- β) stimulates chondrocyte differentiation, while playing a role in inhibiting chondrocyte proliferation, hypertrophy, and mineralization.

This experimental evidence clearly indicates that a complex series of molecular interactions regulate growth in cartilage. In an effort to define the molecular controls of growth at the synchondroses, a cranial base explant culture system was optimized. Using this system, it was determined that hyaluronan and CD44 play a critical role in synchondral growth.

Function perturbing studies demonstrated that hyaluronan and CD44-mediated mechanisms controlled lengthening of the cranial base.

Growth of Cranial Sutures (Fig. 20.11)

Development and growth of human sutures follow a similar pattern to that seen in the rat, with adjustment of timing for length of gestation. The cranial bones appear as initial mesenchymal condensations during the 8th to 12th week of intrauterine life. These condensations begin to mineralize, and expand by radial deposition around the edges of the condensations. At about 14 to 16 weeks of gestation, the cranial bones approximate one another, and suture formation is initiated. While some bone formation continues at these sutures before birth, it is during the postnatal period that the greatest cranial expansion occurs by bone deposition at the sutures.

The presence of the dura mater has been found to be essential for normal development and maintenance

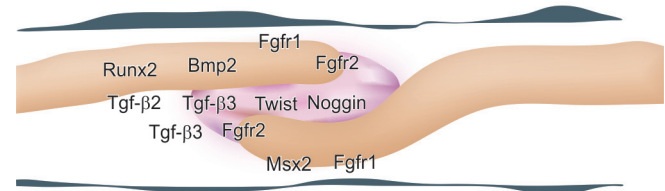


Fig. 20.11: Open suture showing presence of twist, noggin, and Tgf- β 3 in the suture matrix (pink area), Fgfr2 in the edges of the bone fronts, Runx2, Bmp2, Msx2, and Fgfr1 in the bones (brown area spanning the pink gray area), and Fgf2, Tgf- β 2, and Tgf- β 3 in the dura mater (white area below brown and pink regions)

of cranial vault sutures. Cranial suture morphogenesis and the maintenance of cranial sutures as patent bone growth sites are regulated by tissue interactions with the underlying dura mater. Craniosynostosis or premature obliteration of sutures leads to abnormal compensatory morphogenesis throughout the head. Growth factors such as transforming growth factor beta 1 (TGF- β 1), TGF-2, TGF-3, bone morphogenetic protein 2 (BMP2), BMP7, fibroblast growth factor 4 (Fgf4) insulin-like growth factor 1 (Igf-1) and sonic hedgehog (SHH) are found in the suture and the underlying dura mater. Addition of TGF-2 and Fgf-4 to rat or mouse calvaria induces cranial suture fusion, as does removal of TGF-3 activity, similar to the removal of the dura mater. Over expression of transcription factors RUNX2 and MSX2 induces suture obliteration while haploinsufficiency of twist or noggin results in suture obliteration.

The mutations in genes for fibroblast growth factor receptors 1, 2, and 3 (FGFR1, FGFR2, and FGFR3) are associated with craniosynostosis in humans. These are activating mutations, either through constitutive activation pathways, negative regulation of bone growth, repression of developmental genes, or increased affinity for ligand. Mutations in MSX2 and TWIST genes are also associated with human craniosynostosis, but whereas mutated MSX2 undergoes prolonged binding to its DNA binding site, mutations in TWIST result in truncated forms of the protein, resulting in TWIST haploinsufficiency. Many of these gene products interact to regulate expression of one other through tissue interactions between dura mater, bone fronts, and sutures.

Palate Formation

The secondary palate forms as an outgrowth of the maxillary prominence. Interestingly, it was recently shown that sonic hedge hog (SHH) and the FGFR were also expressed in the early palatal epithelium and appear to be induced by FGF10. When this pathway was disrupted in transgenic animals, the palatal processes failed to grow. The normal palatal shelves elevate and grow toward the midline where they fuse and some of the medial edge epithelial (MEE) cells move into the mesenchyme through the epithelial to mesenchymal transitions (EMT) process. The migrating MEE cells can be visualized, if the epithelial cells are bathed in a marker that only penetrates the surface epithelium such as the carboxy 2,7 dichloro-fluorescein diacetate succinimidyl ester (CCFSE). After

the palatal shelves have been organ cultured for 3 days, the label was found in the mesenchymal cells demonstrating the labeled cells had progressed through EMT.

Growth of Facial Sutures

The prenatal expression levels of MSX2, TGF-1, TGF-2, and TGF-3 in transpalatal sutures *in vivo* were similar to those seen in frontonasal sutures. High expression of these mRNAs was seen before birth during the period of suture morphogenesis, and in the postnatal period, during active growth with elongation of the snout. Interestingly, the decline in mRNA expression seen at birth in frontonasal sutures was not seen in transpalatal sutures. One possible explanation for the decline seen in the superficial frontonasal sutures birth trauma as the head is deformed through the birth canal. The transpalatal sutures are protected from the forces exerted on the head at birth because they are deep within the craniofacial tissues near the cranial base. The expression of mRNA and protein levels in transpalatal sutures is more similar to the expression levels noted in coronal and sagittal sutures than those in frontonasal sutures.

Differences in protein expression between the frontonasal sutures and the transpalatal, coronal, and sagittal sutures may reflect different growth patterns and rates of bone growth within the face and head. Most of the bone of the rat cranial vault is laid down postnatally as brain growth is completed before weaning, similar to the rapid postnatal growth of the cranial vault seen in humans. However, the facial bones of both humans and rats grow at a slower rate. Since the palate has to grow rapidly soon after birth to accommodate the teeth, its growth rate would be more similar to the cranial vault growing to accommodate the expanding neurocranium, rather than the slower growing face. Hence, the appearance and distribution of growth factors within the transpalatal sutures would be expected to be more similar to those seen in the coronal and sagittal sutures rather than in frontonasal sutures.

Growth of Condylar Cartilage

The mandibular condylar cartilage (MCC) has a distinctly different developmental and phylogenetic history from the cartilages of the limbs and cranial base. As the cells that divide to effect growth and adaptation in the MCC

are of perichondrial/periosteal rather than chondrogenic origin, the cellular and molecular mechanisms that regulate MCC growth are only beginning to be understood. It has been shown that the potent mitogen fibroblast growth factor 2 (FGF-2) is present in the matrix of the MCC, and that cells of the MCC express cell surface receptors for FGF-2 receptor subtypes.

Insulin-like growth factor 1 (IGF-1), an anabolic factor for matrix synthesis in limb cartilage, has also been reported to be present in the MCC and its type 1 receptor (IGF-1R) has been localized to the chondroprogenitor (prechondroblastic) zone of MCC explants. Less is known of the presence or importance of other growth factors, such as the TGF-beta, PDGF or platelet-derived growth factor, or EGF epidermal growth factor.

With more specific understanding of the characteristics of the "germinal cells" or those which proliferate in the MCC, it might be possible to effect a profound increase in their mitotic capabilities leading to a therapeutic increase in mandibular growth.

Patterning of the Dentition

The hindbrain region of the developing neural tube from which the neural crest migrates is segmented into eight rhombomeres. Segment specific combinatorial HOX gene expression specifies each rhombomeres identity. The migrating neural crest carries this HOX code defined patterning which is transferred to the branchial arches. The HOX code, thus sets up regional diversity within the branchial arch system. It is plausible therefore, that the HOX code of those cells migrating to the tooth forming regions is responsible for specifying and patterning the dentition.

However, the genes are not expressed in region rostral to rhombomeres, which means that no HOX gene expression is seen in the neural crest which migrates to the craniofacial region, including the first branchial arch. In terms of patterning tooth development, we have to look at a subfamily of homeobox genes that show temporal and spatial patterns of expression within the first branchial arch.

Odontogenic Homeobox Code

Based upon such highly specific domains of expression, it has been suggested that these odontogenic homeobox genes provide a homeobox code that specific regions of the developing jaws to assume odontogenic potential.

Various odontogenic homeobox genes identified were:

- MSX genes → MSX-1, MSX-2
- DLX genes → DLX-1, DLX-2
- BARX genes → BARX-1, BARX-2

Each specific region of the homeodomain expresses a unique combination of homeobox genes, which monitor the development of specific teeth. The molecular basis of this patterning is the differential expression of the coded homeobox nuclear proteins which regulate downstream gene transcription. The proteins of this homeodomain act as transcription factor which that result in the activation or inhibition of other genes. These homeobox genes also regulate the expression of other target genes.

MSX Genes

MSX is an important gene involved in tooth formation. MSX stands for *muscle segment homeobox gene*. Mutation of this gene has been associated with facial and dental abnormalities. MSX-1 gene is expressed in migrating neural crest cells and later, in the mesenchymal cells of dental papilla and follicle. MSX-2 genes are involved in signaling interactions, which are essential for the tooth development.

Prior to the initiation of odontogenesis both MSX-1 and MSX-2 exhibit very specific horseshoe-shaped fields of the corresponding mesenchymal expression in the anterior regions of the first arch. These expression patterns are coincident, except along their posterior border where the expression of MSX-1 extends further than MSX-2. This region of isolated mesenchymal MSX-1 expression corresponds to the position of the future primary epithelial thickening. As tooth development progresses, the expression of MSX-1 becomes localized in the mesenchymal cells of the dental follicle and papilla. The domains of expression of MSX-2 also become more restricted to the dental follicle and papilla, but unlike MSX-1, MSX-2, is also expressed strongly in the enamel organ.

DLX Genes

DLX genes are expressed in the migrating neural crest cells and in the first brachial arch. DLX stands for distal-less homeobox gene. The DLX genes have also been conserved during evolution and bear similarity to the distal-less gene of *Drosophila melanogaster*. The

expression of DLX-1 and DLX-2 in the maxillary and the mandibular arch mesenchyme is restricted to the proximal regions where the future molar teeth will develop.

BARX Genes

BARX stands for Bar class Homeobox gene which includes BARX-1 and BARX-2. BARX-1 is homeobox containing transcription factor which exhibits regionalized expression within the ectomesenchyme of the first branchial arch. Bar class homeobox 2 genes (BARX-2) are also a group of homeodomain transcription factors. This group of Homeobox genes was first located in *Drosophila melanogaster* in the locus 11q25. Prior to the appearance of the primary epithelial thickening BARX-1 (along with DLX-2) is expressed in the posterior regions of the first branchial arch mesenchyme, the region of future molar development. There is no BARX-1 expression in the anterior regions. As tooth development proceeds, BARX-1 expression becomes localized exclusively to the mesenchymal regions around the developing molars. Mutation of these genes could be associated with facial and dental anomalies.

PAX Genes

Paired-box homeotic gene (PAX) function by binding enhancer DNA sequences and they modify the transcriptional activity of downstream genes. There are nine PAX genes organized into four groups (Pax1 to Pax9). Of these genes, Pax9 is associated with the development of teeth. Mutations in this gene results in conditions such as hypodontia, transposition, etc. Neubuser et al, found that PAX-9 transcription factor is associated with the genetic mechanism for tooth displacement anomalies, such as palatally displaced canines and canine transposition.

Hedgehog Genes

Sonic hedgehog gene (SHH) is the vertebrate homologue of the *Drosophila* hedgehog gene. SHH is expressed in the epithelial thickenings of the tooth forming regions. SHH along with bone morphogenetic protein (BMP-4) determines the position of future forming tooth germs. SHH is necessary for the initiation of tooth development, epithelial signaling and cuspal morphogenesis. The interaction of SHH gene with other target genes like GLI

is also imperative for tooth formation. GLI Zinc transcription factors are known to act downstream of SHH gene. There are three subtypes namely GLI-1, GLI-2 and GLI-3, which play a vital role in tooth development. Mutant GLI-2 gene results in the formation of abnormal incisors. When GLI-2 and GLI-3 were affected, maxillary incisor development was absent and sizes of mandibular incisors were reduced. When GLI-3 alone was affected, there was no damage in the development of incisors.

Molecular Basis of Hypodontia

Vastardis et al, studied the cause for selective tooth agenesis in human, where mis-sense mutation occurred in the MSX-1 homeodomain. This occurs as a consequence of replacement of arginine with proline amino acid (Arg 196 Pro mutation) in the homeodomain of MSX-1. Tooth agenesis was reported in a family with a ser 105 stop mutation of MSX-1 gene.

Van den Boogard et al, observed a genetic aberration in a Dutch family with tooth agenesis. A stop codon in MSX-1 mutation was identified implying the involvement of this gene in tooth agenesis. Research work by Cobourne on families affected with hypodontia has revealed that it is transmitted as an autosomal dominant disorder with variable expressivity and incomplete penetrance. Missing maxillary laterals and mandibular second premolars have been associated with defects in MSX-1 and MSX-2 genes.

Nieminen found that, a non-sense mutation in the PAX-9 gene was associated with molar tooth agenesis in a Finnish family. The tooth agenesis phenotype involved all the permanent second and third molar and most of the first molars.

Lidral, 2002 concluded that a mutation in MSX-1 gene in chromosome 4 has been identified as the causative factor for oligodontia involving the absence of all second premolar and third molar. Missing first molar and second molars have been linked with a substitution mutation of MSX-1 gene.

With the help of molecular genetics techniques, Peck and Peck, in 2002, assessed a family exhibiting an autosomal dominant trait of missing second premolar and third molars. The affected chromosome was isolated to be in a chromosome 4p and many genes were considered to be responsible for this tooth agenesis. A point mutation was detected in the MSX-1 gene in all affected families. Also, mutation of the PAX-9 transcription

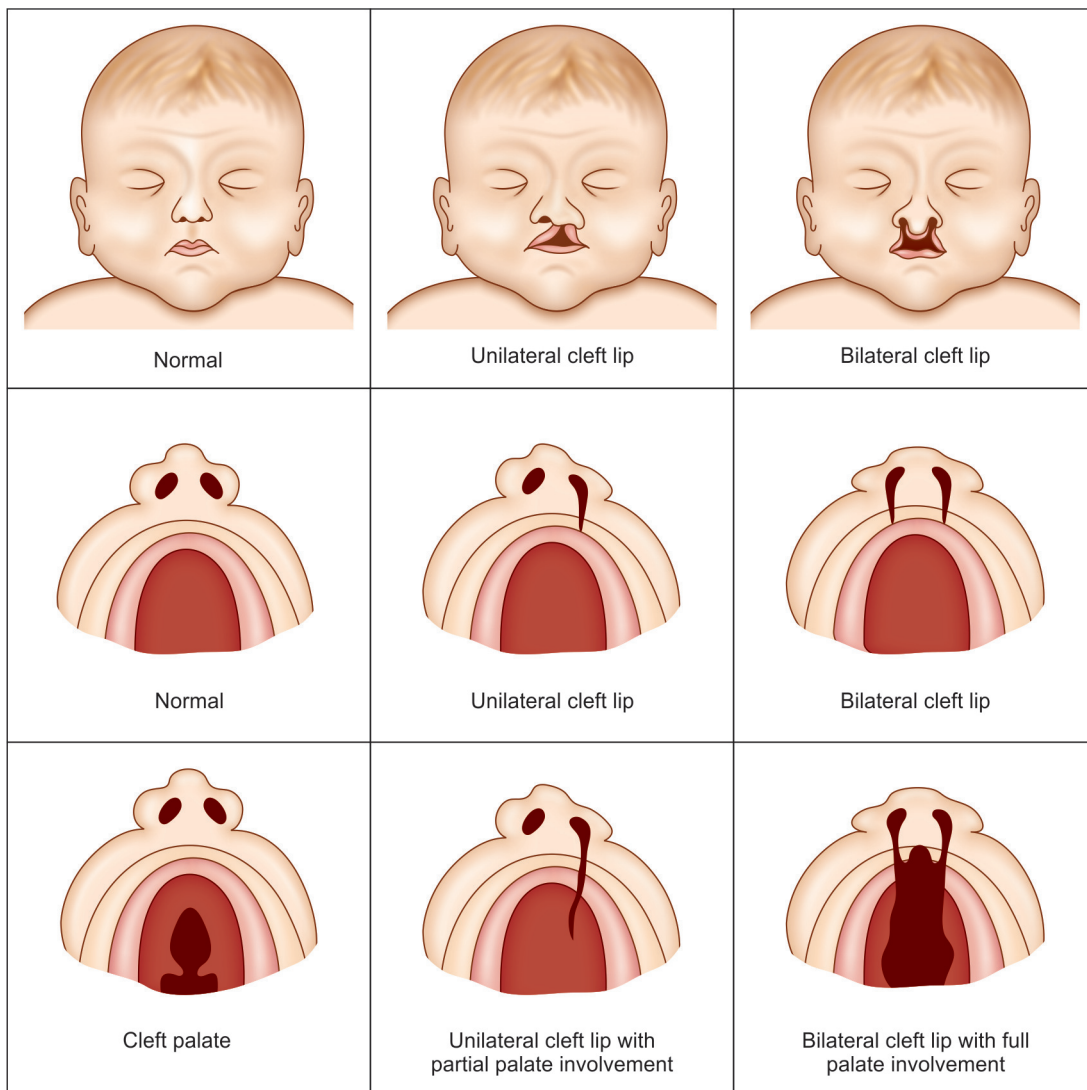


Fig. 20.12: Types of cleft lip and palate

factor has been observed in the familial tooth agenesis and also in case of missing mandibular second premolars and central incisors.

CRANIOFACIAL DEFECTS

Cleft Lip and Cleft Palate (Fig. 20.12 and Tables 20.2 and 20.3)

Cleft lip and cleft palate can be broadly categorized as:

- Non-syndromic CLP/CP
- Syndromic CLP/CP
- Syndromic isolated CP
- Sex-linked CP (CPX)

Non-syndromic CLP/CP

Non-syndromic CLP/CP in humans seems to be etiologically distinctive and still constitute the majority of all classes of clefting disorder.

Various transcription factors and growth factors are involved in non-syndromic cleft lip/cleft palate where mutations in these factors results in the disorder.

Syndromic CLP

Over 300 syndromes are known to have clefting of the lip or palate as an associated feature. As with all clinically recognizable syndromes, cases of syndromic CLP or CP

Table 20.2: Growth factors involved in CL/CP

Genes	Loci
Transforming Growth Factor α (TGF α)	2p11-13
Transforming Growth Factor β (TGF β)	14q23-24
Retinoic Acid Receptor Alpha (RARA)	17q21
GABA Receptor β 3 (GABRB3)	15q11.2-12
B-cell Leukemia/Lymphoma (3 BCL3)	19q13
Jagged 2 (Jagg 2)	14q32
Apolipoprotein C II (APOC2)	19q13.1

Table 20.3: Transcription factors involved in CL/CP

Genes	Loci
<i>Homeobox genes</i>	
Muscle Segment (MSX1)	4p16.1
Lim Homeobox (LHX8)	4q25-31
Bar class (BARX)	11q25
Distal less (DLX2)	2q32
<i>Other genes</i>	
Endothelin 1	6p23-24
Glutamate Decarboxylase (GAD 67)	2q31

can be broadly subdivided into—(i) those that occur as part of characterized Mendelian disorder (single gene defects); (ii) those arising from structural abnormalities of the chromosomes; (iii) Syndromes associated with known teratogens; (iv) those whose causation remains obscure and are therefore, currently uncharacterized.

One of the most common human autosomal dominant disorders associated with CLP is van der Woude syndrome. Twin studies revealed that a non-sense mutation in the interferon regulatory factor-6 (IRF6) gene resulted in van der Woude syndrome.

Some of the syndromes associated with CLP are, Pierre Robin syndrome, CLP-ectodermal dysplasia syndrome (CLPED-1), ectrodactyly, ectodermal dysplasia, orofacial cleft (EEC syndrome).

Syndromic CP

In addition to syndromic CLP, progress has also been made in elucidating the genetic mechanisms behind several syndromic causes of isolated CP. Some of the syndromes associated with CP are—mandibulofacial

dysostosis (Treacher Collins syndrome), holoprosencephaly, type-3 Stickler syndrome.

Sex-linked CP (CPX)

Philip Stainer and Gudrun Moore found the Sex (X) chromosome linked form of cleft palate (CPX) and an associated disorder ankyloglossia can occur due to mutations in a particular gene T-Box 22. T-Box genes are members of a family of transcription regulators that share a common DNA-binding domain, the T-Box.

Inheritance Patterns of Cleft Lip and Palate

Monogenic or single gene disorders: Approximately half of the recognized syndromes associated with cleft lip and palate are due to single gene disorders with equal distribution between autosomal dominant and autosomal recessive. Single gene defect may give rise to Mendelian pattern of inheritance, either of isolated cleft lip (palate) or in multiple malformations associated with cleft lip with or without cleft palate.

Polygenic or multifactorial inheritance: Several genes, each with a relatively small effect, act in concert with poorly defined environmental triggering mechanisms leading to the expression of the abnormality. Thus, such cases show a slight familial tendency but do not conform to simple Mendelian inheritance patterns.

Chromosomal abnormalities: Chromosomal abnormalities account for 18 percent of the clefting syndromes and would invariably be associated with other malformations, delayed development and poor prognosis. Chromosomal abnormalities, notably trisomy D and also, less frequently trisomy E, may cause multiple malformations including cleft lip (palate).

Familial: Fogh-Anderson's family studies showed that siblings of patient with cleft lip had increased frequency of cleft lip and cleft palate, but no increased frequency of cleft palate alone. Siblings of patients with cleft palate had increased frequency of cleft palate, but not CL and CP.

Sex predominance: More males are born with cleft lip and cleft palate than females and more females than males have cleft palate alone.

Racial incidence: The incidence of cleft lip and cleft palate is greatest in the Mongoloid population, being greater than that in the Caucasian population, which is in turn

greater than in the Negroid population. In contrast, the racial differences for cleft palate alone are not significant.

Craniofacial Syndromes

A syndrome is recognized to represent multiple malformations occurring in embryonically non-contiguous areas. Some of the syndromes with dental importance are:

- Crouzon's syndrome
- Apert's syndrome
- Treacher Collins syndrome
- Pfeiffer syndrome
- DiGeorge syndrome, velocardiofacial syndrome, conotruncal anomalies face syndrome (CATCH 22 spectrum).

Crouzon's Syndrome

It is a frequent form of craniofacial dysostosis, characterized by multiple anomalies of the craniofacial skeleton with an autosomal dominance inheritance pattern. Its manifestations are usually less severe than the Apert's syndrome and there are no malformations of the extremities. Characteristic premature synostosis of both coronal sutures results, with a resultant brachycephalic shape to the skull, midface hypoplasia with an Angle's class III malocclusion, hypoplastic orbits with a proptosis, Parrot beak nose and short anterior cranial base.

Genetic etiology: It is caused by multiple mutations in the fibroblast growth factor receptor2 gene (FGFR2). Mutation in tyrosine kinase receptor, at IgII-IgIII domain. Crouzon's with acanthosis nigricans has been described with a specific Ala391Glu mutation in FGFR3. More recently, Muenke and co-workers reported that it is due to an amino acid substitution (Pro250Arg) that results from a single point mutation in FGFR3 on chromosome 4P. This new syndrome called FGFR3 syndrome and the associated coronal synostosis syndrome may present as bilateral coronal synostosis with minimal midface involvement.

Apert's Syndrome

Apert's syndrome (also known as Apert-Crouzon disease) is characterized by skull malformation (acrocephaly of brachysphenocephalic type) and syndactyly of the hands and feet of a special type (complete distal fusion with a tendency to fusion also of the bony structures). It is

associated with an autosomal dominant inheritance pattern.

Genetic etiology: At the molecular level, one of the two fibroblast growth factors 2 gene (FGFR2) mutations involving amino acids (ser 252 trp and pro 253 Arg) are found to cause Apert's syndrome. Tyrosine kinase receptor is affected at the extracellular IgII-IgIII domain. In Apert's syndrome, the associated midface hypoplasia is thought to be secondary to a cartilage maturation defect affecting the cranial base.

Treacher-Collins Syndrome

Treacher Collins syndrome, or mandibulofacial dysostosis, is an autosomal dominant condition with variable expressivity. It is generally characterized by bilaterally symmetrical abnormalities of the structures within the first and second branchial arches. It is characterized by malar hypoplasia, mandibular hypoplasia, downward palpebral fissures and coloboma of lower eye lid and malformed external ears.

Genetic etiology: The gene for Treacher Collins syndrome has been mapped to chromosome 5q31.3-q33.3. The Treacher Collins Syndrome gene resides between the colony-stimulating factor receptor (CSFR) gene and the osteorectin (SPARC) gene, a region of less than 1 million base pairs of DNA on chromosome 5.

Theories of pathogenesis include the failure of differentiation of the branchial arch mesoderm, defective facial bone ossification, and tissue ischemia resulting from stapodial artery hypoplasia. Craig et al, suggested that variability in the extent of the deformities with this condition is due to the influence of "strong" or "weak" gene acting at an earlier or later period of the embryo's development. Behrents et al, pointed out that all major aspects of MFD are fully expressed by the 15th week of embryonic development. Current research suggests that the abnormality may occur early as developmental defects of the neural crest cells.

Pfeiffer Syndrome

Features of Pfeiffer syndrome include craniostenosis, orbital dystopia, midface hypoplasia, broad and medial deviated thumbs and great toes and partial soft tissue syndactyly of the hands and feet. Pfeiffer syndrome is said to have an autosomal dominance inheritance pattern.

Genetic etiology: It is heterogeneous because it is caused by a single recurring mutation of the FGFR1 gene and by several different mutations affecting the FGFR2 gene.

DiGeorge Syndrome

DiGeorge syndrome, velocardiofacial syndrome and conotruncal anomalies face syndromes come under disorders in CATCH 22 spectrum. CATCH 22 is due to the deletion of long arm of chromosome 22 (more specifically, foci 22q11). Cardiac defects, abnormal facies, thymic hypoplasia, cleft palate and hypocalcemia are the clinical features. The origin of defects is caused by abnormal development of neural crest cells which contribute to the formation of all the affected structures.

FUTURE OF MOLECULAR RESEARCH IN CRANIOFACIAL GROWTH

Significant advances have been made in understanding how various tissues and factors interact to regulate suture patency. We are now beginning to determine which tissues and factors could be used to either prevent, or delay suture fusion, potentially as treatment for craniosynostosis or poorly growing sutures. Several studies have reported the ability of various factors or antibodies of rescuing sutures from obliteration. *In vitro* culture of fetal rat and mouse calvaria have shown that both TGF-3 and neutralizing antibodies to TGF-2 can be used to rescue coronal sutures from obliteration. Once the biology of the suture response to growth factor is better understood, it will be possible to apply these factors to human subjects with premature suture obliteration.

The growth factors and related molecules present in the mandibular condylar cartilage (MCC) are being accessed. Some genes, like IHH, have been shown to be upregulated when active condylar growth ensues. Improved understanding of the molecular and biochemical processes in the condylar cartilage open the possibility of "growing the mandible".

Adult stem cells play an important role in the remodeling and the repair of tissues throughout the life of an organism. Recent research shows that adult stem cells are capable of giving rise to multiple cell types produced from different germ layers. Adult stem cells have been identified from dental pulp, periodontal ligament, jaw bones, etc. Complete discovery of the molecular mechanisms that regulate stem cell behavior

might simplify the treatment of dento-facial anomalies. Stem cells can be cultured to replace cells or tissues lost by trauma or disease.

In gene therapy, a "corrected" gene is inserted into the genome to replace an "abnormal," disease-causing gene. A carrier called a vector must be used to deliver the therapeutic gene to the patient's target cells. Currently, the most common type of vectors are viruses that have been genetically altered to carry normal human DNA. Viruses have evolved a way of encapsulating and delivering their genes to human cells in a pathogenic manner. Scientists have tried to harness this ability by manipulating the viral genome to remove disease-causing genes and insert therapeutic ones. Target cells such as the patient's liver or lung cells are infected with the vector. The vector then unloads its genetic material containing the therapeutic human gene into the target cell. The generation of a functional protein product from the therapeutic gene restores the target cell to a normal state. Gene therapy is still in infancy but is full of promise.

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Emotional Growth

CHAPTER OUTLINE

- Schools of Psychology
- The Dynamics of Emotional Development from Infant to Adult
- Theories of Emotional Development
 - Psychoanalytical theory
 - Psychosocial theory
 - Cognitive theory
 - Classical conditioning theory
 - Operant conditioning theory
 - Social learning theory
 - Hierarchy of needs
 - Psycho-orthodontic theory
- Habit Intervention and Emotional Growth
- The Role of Malocclusion in Psychological Development
 - Self concepts
 - Emotional development and orthodontic treatment need
 - Treatment during preadolescence or adolescence
- Emotional Development and its Relation to Cooperation in Treatment

Knowledge of a child's development includes not only the study of the child's biological development but also of the emotional and cognitive development. At each stage, there are certain kinds of problems to be solved and when the child succeeds, he can and will go on to tackle new problems and to grow through solving them. Knowledge of the mental and emotional growth process helps the practicing orthodontist, pedodontist and maxillofacial surgeon to deal more effectively with their patients and improve patient compliances. "Emotional development" refers to the child's intrapsychic growth as reflected by his interpersonal behavior. Initially, an infant is interested only in self, intent only on pursuing his own physical comfort. As he grows older, he becomes

aware of external forces and people and by the end of first year, will develop a sense of reassurance and trust in those who he learns are available consistently to respond to his needs. "Cognitive development" may be thought of as the development of intellectual process.

Facial esthetics has been found to be a significant determinant of self and social perceptions and attributions. The perception of facial esthetics influences psychological development from early childhood to adulthood.

Salzmann, 1967, included in his definition of need for orthodontic care, the effects of dentofacial handicaps on the functional, esthetic, and personality development of children in addition to the usual quantitative measures of malocclusion. Because orthodontic treatment will alter the esthetic appreciation of the total self, such intervention will affect interpersonal growth in the child, as well as in the development of one's self-image.

Thus, it is essential for the orthodontist to know about the normal emotional growth of the child for the purpose of providing better treatment results.

SCHOOLS OF PSYCHOLOGY

The earliest schools were the structuralist and functionalist schools. The goal of the *structuralist school* was to determine the structure and dimensions of the consciousness. They used introspection as their tool. The *functionalist school* deals with how a man adjusts to the environment and how he might change his adjustments and improve the methods of dealing with the environment. As the field of psychology developed, the other schools of thought emerged. The *behaviorist school* believes that the study of behavior should be limited

to what can be directly observed or manipulated, that is, overt behavior. BF Skinner and Pavlov were strong advocates of this concept. *Gestalt school* stated that the real understanding of behavior could be made only through total observation and studying the total situation or gestalt. It was against breaking the human behavior into different elements and emphasis was on studying the person as a whole. In 1900's, Sigmund Freud, a Viennese Physician, proposed a theory which shocked the world first. It became a very influential theory and caught the attention of the whole world. Freud stated that sex is the fundamental drive that causes certain behavior patterns and further added that the influence of sex was present in many unconscious behavior patterns also. It is called as the Psychoanalytical theory.

THE DYNAMICS OF EMOTIONAL DEVELOPMENT FROM INFANT TO ADULT

The emotional, cognitive and social changes that take place in a human being can be studied under the following stages:

- i. Newborn to first three years of life;
- ii. Three years to six years of life (Primary dentition years);
- iii. Six years to twelve years (transitional years);
- iv. After 12 years (adolescence).

Newborn to Three Years of Life

The development of child from conception to age three years marks the most dramatic years in terms of growth and development and to a certain extent, the emotional development.

Emotional Changes

There are different emotions like shame, guilt, joy, fear, anger and sadness. Emotions can be assessed by measuring physiologic responses like heart rate or by person's thoughts like feeling of depression or by observing behavioral responses. In assessing the emotional state of the children, the first two methods are of little use. As a general rule, in the first year of a child's life, adults assign to the child whatever emotions they believe that the child should feel in a particular situation. When a child spills milk, each parent may interpret the response in different ways, like frustration, guilt, fear and so on.

Pinkham believes that there appears to be an awakening of emotional status within the child between 4 months and 10 months of age. Mussen and others noted that the infants were capable of displaying behavior like anger, frustration. Sadness on separation from a parent, joy on reunion, and jealousy of peers and siblings become evident as a child approaches his or her first birthday. Uncertainty and certainty are a pair of elements that emerge in infancy. This leads to fear or lack of fear. If the child experiences any startling situation, the child may develop fear if it is not able to understand the situation. Avoiding startling situation will help children to react well to new environmental situations.

Emotionally, the infant, during the first few weeks of life to 4 months, seems to exist only for himself in a totally self-centered fashion accepting his dependency upon the mother and offering nothing in return. Thus, the self-centered organism becomes aware that need fulfillment is coming from outside oneself, without yet knowing what 'outsideness' is. Thus, the child experiences the self and caretaker as one. This phase of development is called the *symbiotic phase*. It will last until 10 months of age, when the separation and individualization will begin. Fear of strangers is a universal finding after 7 to 10 months of age. Fear of separation is another fear which develops around 6 months of age, peaks between 13 and 18 months of life and then declines. The basis for this fear is probably the result of developing remembrance of the parent even in their absence which correlates with object permanence. Most children overcome this problem of separation anxiety by 36 to 40 months of age. Parental care is very critical during this stage of emotional development. Ainsworth concluded that children who have strong relationships with their primary caregivers could utilize the relationship from which they could venture into wider social circles by exploration. On the contrary, children with poorly developed relationships with their caregivers are not able to undertake such exploration because they lack sense of security.

Cognitive Changes

Unlike other mammals, human infants are totally dependent upon another person for survival during a significant period of early childhood. This dependency not only includes physical care but also emotional and cognitive needs. Many psychologists now recognize that

there is cognitive ability in the newborn. There is evidence that newborns experience sensations of pain, touch and changes in position. There is also evidence of the newborns ability to smell, see and hear. Mussen and coworkers, 1984, stated that there are four important areas of cognitive development during the first year of life: (i) Area of perception—which states that even very young infants have the ability to perceive movement, facial relationships and color. (ii) Recognition of information—infants can recognize certain stimuli such as face when viewed from various angles. This allows a child to recognize the similarity of new objects compared with older ones because of their ability to generalize on the crucial elements. (iii) Ability to recognize—children can group things together by way of their shape, color and use by the age of one year. (iv) Enhancement of memory is the fourth cognitive development of the first year of life. Very young infants as old as 6 months of age have the ability to recall past experiences.

In the study of cognition in infants two theories are commonly accepted. First is the learning theory. Both classic and operant conditioning come into play. Sucking the nipple or feeding combined with lullaby is an example for *classical conditioning*. Giving a reward for good behavior is an example of *operant* or *instrumental conditioning*.

Piaget's cognitive development is the second theory which states that much of the intellectual development of the child from birth to two years of life is the result of the child with the objects in the environment. Piaget believes that the child should develop knowledge in the following three areas:

Object permanence: Objects continue to exist to the child even when they are not perceived by the child.

Causality: Objects have uses, and events have causes. Piaget used the term circular reaction to describe the changes that occur in this area. Accordingly he says there are three types of circular reactions. Primary circular reaction explains an already known satisfying action like thumb sucking. A secondary circular reaction is the recreating of an accidentally discovered cause and effect. Tertiary circular reactions involve experimentation.

Symbolic play: One object can represent another.

Language development of the infant is very slow initially. Mean expressive vocabulary of an eighteen

month old child is 10 words. Levine et al. noted that by three years of age this increases to 1000 words.

Social Changes

First year: Child is completely dependent on the parents. For first few months child does not show clear differentiation among people. Nonreflexive smiling starts at 2 to 3 months. This is the first major social behavior of the infant excluding crying.

Second Year: Second year of development marks great social progress. Role model development begins to develop and continues for years to come. Children who see nonaggressive ways of handling are likely to acquire that approach. Parental affection and verbal communication should be maintained at this stage. Discipline should be educational and not physical. Physical punishment will make the child behave worse.

Third year: Child starts to eat independently. Toilet training takes place in this period. This period represents the terrible two's stage of development.

Three Years to Six Years of Life

This age group with their skill at talking to people and relating to them is one that is delightful.

Emotional Changes

The fears of strangers, separation from parents and also to new experiences would have been diminished by the beginning of this stage. The process of self-control and control of emotions like frustration and fear develop between the ages of 3 and 6. Development of self-control is the most important emotional change during this stage. Inability to exert self-control by the child leads to aggression. There are two kinds of aggression: *Instrumental aggression* designed to achieve a goal such as taking a piece of candy from a sibling; *hostile aggression* is intended to cause pain to another person. Parental behavior that is inconsistent and unclear in enforcing rules leads to aggression in children.

This is also the period when a sense of *ambivalence*, that is love and hate for important people in ones life, is felt. This is brought about by the on-again, off-again fulfillment of the child's desires by the caretaker. Ability or inability to separate from the primary caretaker and to relate well with other people will be forever important

stage of the adequacy of completion of this early phase of personality development.

The child's sense of sexual identity emerges and the child also acquires a certain amount of masculine or feminine quality. A sense of identity and self-esteem also develop.

The child, at the end of its sixth year, is not emotionally mature but is emotionally complex. The child is capable of friendship/hostility, self-control/aggression, guilt/anxiety and is susceptible to praise and also hurt feelings.

Cognitive Changes

This period represents an enormous cognitive change. The child's power of questioning and reasoning grows substantially. The child begins to question with "How" and "Where" from simple "Why". The child's mental prowess develops rapidly and it acquires the ability to think symbolically with imaginations. However, the child's imaginations are still not sophisticated. The preconceptual mind is also centered. Late in this period, the child begins to acquire reading and writing skills. There is increased vocabulary, attention capacity rises, toleration of separation from parents and increased control of impulses.

Social Changes

During this stage, the child learns to play simple team games and cooperative play is possible. Value system and self-discipline on basic urges develops and a consciousness that enables the child to feel guilt, emerges. There is a dramatic psychosocial transition that takes place in this age group. Shonkoff pointed out that the fantasies, the preschoolers enact out, are rich in relation to sexual and adult values.

Six Years to Twelve Years of Life

The orthodontist must provide answers to both parents and the child about their appearances of the child, intercept the developing malocclusions and take care of the emotional changes that could take place because of malocclusion during this stage.

Emotional Changes

Crying, tantrums present in younger children will be relinquished by this stage. The child will be focused

towards his needs and likes doing home work, caring for pets, team sports etc. The need for parents to direct the child's attention recedes and by the age of 12, the child's ledger of wants and desires increases. The body image starts to become an emotional feature in this stage and becomes dramatic in adolescence. The facial appearance of the child counts a lot in further emotional development of the child. Teasing by peers may exacerbate the problem. Majority of the children find overall emotional satisfaction only when they are accepted well socially by their friends. Lack of acceptance and teasing can be damaging emotionally. Child should be channelized to handle and recover from humiliation, frustration, loss and disappointment. Failing to do so presents a real danger in adolescence.

Cognitive Changes

Mental capacity grows extensively. White, in 1968, stated that between the ages of 5 and 7 years, a reorganization of central nervous system takes place and this accounts for the dramatic increased ability of the child to remain diligent or attentive to a problem. By age 12, most children have the sophisticated ability to produce oral and written communications. By the age of 12, a child will be able to assimilate majority of information.

Social Changes

This is a complicated phase when compared to the previous two stages because of school, the increasing importance of friends and also because of the increase in the child's social environment. Most children accept school positively and remain enthusiastic about school experiences. Teachers play an important role in the socialization of the child. The behavior of the child could be affected by the peer group he or she joins. This stage marks the advent of stronger, stable and meaningful friendships. Usually friendships are made with the same sex.

From 12 Years to 18 Years (Adolescence)

Adolescence represents an extremely important time in the emotional and social development of an individual. Adolescence is a psychological state of maturation while puberty is a physical state of maturation. During this period, a wide difference in the level of psychological maturation develops.

Emotional Changes

Rapid and dramatic changes occur in adolescence, both physically and emotionally. The self-confidence and personal identity of an individual will be jeopardized if his or her feelings about their body image are negative. Mussen pointed out the important issues which create anxiety for this group. They are:

- Being attractive or unattractive
- Being loved or unloved
- Being strong or weak
- Being masculine or feminine.

Onset of menstruation may provoke anxiety in females. The advent of puberty and hormonal changes lead to sexual feelings and urges. Family guidance, peer group values and the individual's own values are the factors which determine how an individual will deal with the changes.

The last important emotion during this stage is love. Adolescence is a period where there is possibility of commitments and relationships. Broken relationships can lead to depression for the affected individual.

Cognitive Changes

Cognitive development continues through adolescence and by the end of late adolescence the individual is capable of performing extremely sophisticated intellectual tasks. Ability to store information and formal operational thinking are the highlights of the maturation of cognitive capacity of adolescents. The thoughts of adolescents are both, analytic and introspective. Many of the adolescents mature into skillful enthusiastic communicators; many become opinionated and argumentative. These factors make for the parents' and teachers' difficult times.

Social Changes

Socially, adolescence represents the final transition from childhood to adulthood. The successfully emerging young adult will be able to establish and maintain loving and sexual relationship with partners, be independent of parents, will be self-directed, and cope up with peers. Failure will lead to alcoholism, drug dependence, drop-outs from school, suicidal tendencies and so on.

During adolescence there is an increase in the size and range of acquaintances. Adolescents also like to be popular among their group. The desirable qualities of an adolescent are:

Friendly like other people, energetic, enthusiastic, flexible and forgiving, good sense of humor, tolerance, self confident with leadership quality, appears natural, outgoing, not conceited, make others feel good. Thus by the end of adolescence the child develops a sense of identity and true resolution.

Age Eighteen to Death (Adulthood)

Setting aside chronology, a true adult is the stabilized person who has emerged from the previous stages as responsible for the rest of his own life and that of the next generation. Normal adult is vulnerable to uncomfortable feelings of shame, related to lack of satisfaction with himself in relationship to himself and others, guilt in relation to the demands of superego, depression in relationship to loss, irrational fears and anxieties, and conflicts relating to love and hate and masculinity and femininity. However, he has an ego that is strong enough to judge, decide and cope in the face of these assaults.

Young and middle adulthood is a time of equal give and take between peers, and more give to children and older adults. Until his last day, the strength of ego will be tested constantly in small ways and sometimes, in monumentally large ones.

THEORIES OF EMOTIONAL DEVELOPMENT

G. Stanley Hall (1846-1924) is recognized as the founder of Emotional Development and Psychology. He can easily be called the founder of organized psychology as a science and profession and the father of the child study movement. He stated that "Theories are nothing more than a set of Concepts and Propositions that allow the Theorist to describe and explain some aspects of experience". It helps to explain various patterns of behavior and emotions. Developmental psychology is concerned not only with describing the characteristics of psychological change over time, but also seeks to explain the principles and internal workings underlying these changes.

During the 17th and 18th century, philosophers stated that children are inherently bad or good. However in the 19th century, theorists noted that positive or negative activity of character depends on child's experiences. Development involves two processes—maturity and learning. Maturity means the changes which depend on innate abilities. Learning involves the ability to absorb

from and interact with the environment. "Learning Theory" is a discipline of psychology that attempts to explain how an organism learns. It consists of many different theories of learning, including instincts, social facilitation, observation, formal teaching, memory, mimicry, and classical and operant conditioning. The various theories of psychology are given below.

Theories of Psychology

- Psychoanalytical theory by Sigmund Freud
- Psycho-social theory by Erik Erickson
- Cognitive theory by Jean Piaget
- Behavior learning:
 - Classical conditioning by Pavlov
 - Operant conditioning by BF Skinner
 - Observational learning by Bandura.

The Psychoanalytic Theory (Sigmund Freud)

Sigmund Freud (1856-1939), was an Austrian doctor who revolutionized ideas on how the human mind works. Freud established the theory that the unconscious motives control much behavior of an individual. He, thus, greatly advanced the fields of psychiatry and psychology. The conscious mind is what you are aware of at any particular moment, your present perceptions, memories, thoughts, fantasies and feelings. Working closely with the conscious mind is what Freud called the preconscious, or what we might today call "available memory"—anything that can easily be made conscious,

the memories you are not at the moment thinking about but can readily bring to mind. There is no problem with these two layers of mind. But Freud suggested that these are the smallest parts! The largest part by far is the unconscious. It includes all the things that are not easily available to awareness, including many things that have their origins there, such as our drives or instincts, and things that are put there because we can't bear to look at them, such as the memories and emotions associated with trauma. According to Freud, the unconscious is the source of our motivations, whether they are simple desires for food or sex, neurotic compulsions, or the motives of an artist or scientist. And yet, we are often driven to deny or resist becoming conscious of these motives, and they are often available to us only in disguised form.

Freud hypothesized three systems in the theory of the understanding of the intrapsychic process and personality development. Personality of an individual is based on the interaction between three systems within each individual. He called the systems—Id, ego and super ego (Figs 21.2 to 21.3).

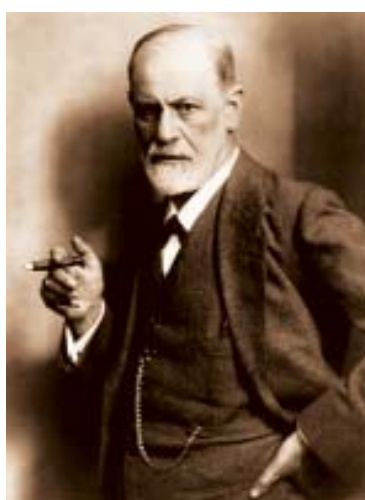


Fig. 21.1: Sigmund Freud (1856-1939)

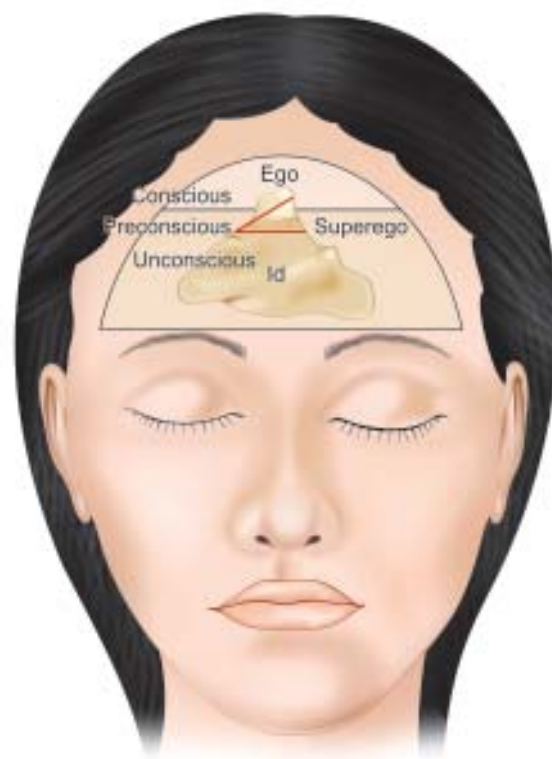


Fig. 21.2: Interaction of id, ego, superego at various levels of mind

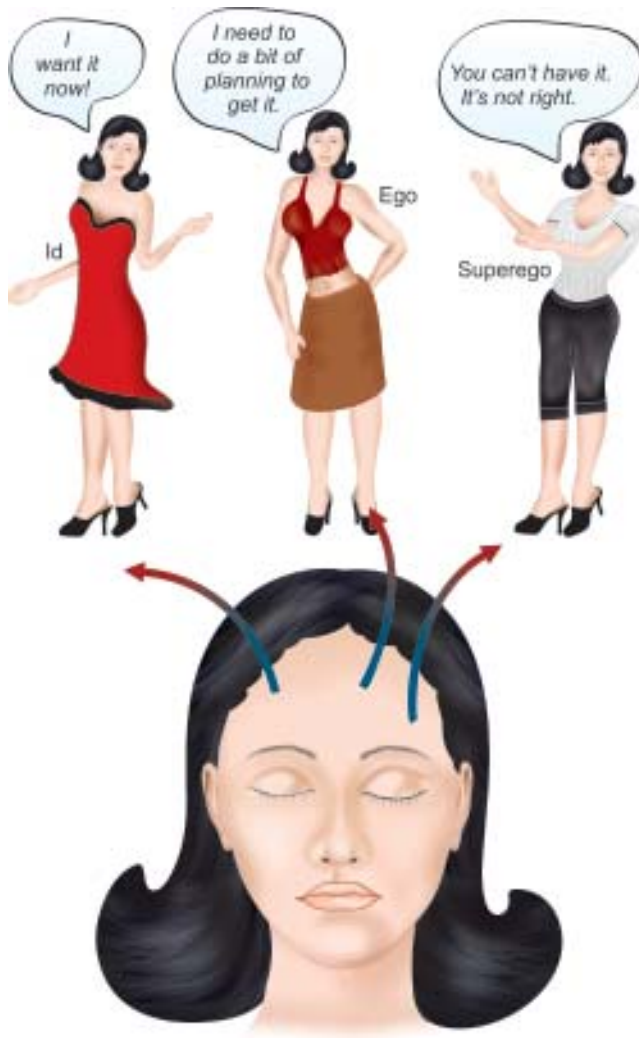


Fig. 21.3: Diagram showing the differences in thinking of id, ego and superego

The Id/Fantasy

Freud believed that the id represented unregulated instinctual drives and energies striving to meet bodily needs and desires. They operate on pleasure principle. He recognized that each person is born with various natural drives that he called instincts, such as the need to satisfy sexual desires and the need to be aggressive. The id is the source of such instincts. The drives are necessary for the survival of the species through procreation and self-defense. The inner urges of the id can find satisfaction only in external sources. It aims at immediate satisfaction of libidinal urges. It is immoral, illogical and lacks unity of purpose.

The Ego/Reality

It is described as that part of the self concerned with the overall functioning and organization of the personality through the ego's capacity to test reality, the utilization of ego defense mechanisms and of other ego functions such as memory, language, intelligence, and creativity. Thus, ego is concerned with maintaining a stage in which an adequate expression of id drives and satisfaction can occur within the constraints of reality and the demands and restrictions of the superego. Ego serves as the mediating or integrating part of personality.

Gabriel (AJO 1993) showed low ego strength to be predictive of high compliance in prepubertal children, but predictive of low compliance in adolescents.

Superego/Idealistic

The superego is a person's conscience. A person's ideas of right and wrong—learned from parents, teachers, and other people in authority become part of the person's superego. The superego is derived from familial and cultural restrictions placed upon the growing child. Freud hypothesized that superego functions were derived from the struggle over strong feelings of the child. The superego stems from the internalization of the feelings of good and bad, love and hate, praising and forbidding, reward and punishment.

Thus, superego holds the id in check and makes value judgments about the individual urges and impulses.

All people have some conflict among the three parts of the mind, but certain people have more conflict than others. For example, the superego might oppose angry behavior. In that case, the id and the superego would clash. If the parts of the mind strongly oppose one another, psychological disturbances result.

Defense Mechanisms

The ego has a very stressful job. It must mediate between the impulsive id, the conscientious superego, and the pressures of the outside world. Freud believed that in order to relieve some of this stress, we act out in the form of defense mechanisms. Many personality disorders are because of the conflict between ego and superego. Ego has a difficult time satisfying both the id and the superego, but it does not have to do so without help. The ego has some tools it can use in its job as the mediator, tools that help defend the ego. These are called defense mechanisms or the defenses. When the ego has

a difficult time making both the id and the superego happy, it will employ one or more of these defenses. Defense mechanisms are unconscious responses that an individual makes in an attempt to cope with and reduce anxiety.

The different types of defense mechanisms are as follows:

- *Denial* involves blocking external events from awareness. If some situation is just too much to handle, the person refuses to experience it. As you might imagine, this is a primitive and dangerous defense—no one disregards reality and gets away with it for long! It can operate by itself or, more commonly, in combination with other, more subtle mechanisms that support it.
- *Repression*, also called "motivated forgetting," is just that: not being able to recall a threatening situation, person, or event. This, too, is dangerous, and is a part of most other defenses.
- *Asceticism*, or the renunciation of needs, is one most people haven't heard of, but it has become relevant again today with the emergence of the disorder called anorexia. Preadolescents, when they feel threatened by their emerging sexual desires, may unconsciously try to protect themselves by denying, not only their sexual desires, but all desires. They get involved in some kind of ascetic (monk-like) lifestyle wherein they renounce their interests in what other people enjoy.
- *Isolation* (sometimes called intellectualization) involves stripping the emotion from a difficult memory or threatening impulse. A person may, in a very cavalier manner, acknowledge that they had been abused as a child, or may show a purely intellectual curiosity in their newly discovered sexual orientation. Something that should be a big deal, is treated as if it were not.
- *Displacement* is the redirection of an impulse onto a substitute target. If the impulse, the desire, is okay with you, but the person you direct that desire towards is too threatening, you can displace to someone or something that can serve as a symbolic substitute.
- *Turning against the self* is a very special form of displacement, where the person becomes their own substitute target. It is normally used in reference to hatred, anger, and aggression, rather than more positive impulses, and it is the Freudian explanation for many of our feelings of inferiority, guilt, and depression.
- *Projection*, which Anna Freud also called displacement outward, is almost the complete opposite of turning against the self. It involves the tendency to see your own unacceptable desires in other people. In other words, the desires are still there, but they are not your desires anymore.
- *Altruistic surrender* is a form of projection that at first glance looks like its opposite: Here, the person attempts to fulfill his or her own needs vicariously, through other people.
- *Reaction formation*, which Anna Freud called "believing the opposite," is changing an unacceptable impulse into its opposite. So a child, angry at his or her mother, may become overly concerned with her and rather dramatically shower her with affection. An abused child may run to the abusing parent.
- *Undoing* involves "magical" gestures or rituals that are meant to cancel out unpleasant thoughts or feelings after they've already occurred. Anna Freud mentions, for example, a boy who would recite the alphabet backwards whenever he had a sexual thought, or turn around and spit whenever meeting another boy who shared his passion for masturbation.
- *Introjection*, sometimes called identification, involves taking into your own personality characteristics of someone else, because doing so solves some emotional difficulty. For example, a child who is left alone frequently, may in some way try to become "mom" in order to lessen his or her fears. You can sometimes catch them telling their dolls or animals not to be afraid. And we find the older child or teenager imitating his or her favorite star, musician, or sports hero in an effort to establish an identity.
- *Regression* is a movement back in psychological time when one is faced with stress. When we are troubled or frightened, our behaviors often become more childish or primitive. A child may begin to suck their thumb again or wet the bed when they need to spend some time in the hospital. Teenagers may giggle uncontrollably when introduced into a social situation involving the opposite sex.
- *Rationalization* is the cognitive distortion of "the facts" to make an event or an impulse less threatening. We do it often enough on a fairly conscious level when we provide ourselves with excuses. But for many

people, with sensitive egos, making excuses comes so easy that they never are truly aware of it. In other words, many of us are quite prepared to believe our lies.

- *Sublimation* is the transforming of an unacceptable impulse, whether it be sex, anger, fear, or whatever, into a socially acceptable, even productive form. So someone with a great deal of hostility may become a hunter, a butcher, a football player, or a mercenary. Someone suffering from a great deal of anxiety in a confusing world may become an organizer, a businessperson, or a scientist. Someone with powerful sexual desires may become an artist, a photographer, or a novelist, and so on. For Freud, in fact, all positive, creative activities were sublimations, and predominantly of the sex drive.

Stages of Psychoanalytical Theory

Freud advanced a theory of personality development that centered on the effects of the sexual pleasure drive on the individual psyche. At particular points in the developmental process, he claimed, a single body part is particularly sensitive to sexual, erotic stimulation. These *erogenous zones* are the mouth, the anus, and the genital region. The child's libido centers on behavior affecting the primary erogenous zone of his age; he cannot focus on the primary erogenous zone of the next stage without resolving the developmental conflict of the immediate one.

A child at a given stage of development has certain needs and demands, such as the need of the infant to nurse. Frustration occurs when these needs are not met; Overindulgence stems from such an ample meeting of these needs that the child is reluctant to progress beyond the stage. Both, frustration and overindulgence, lock some amount of the child's libido permanently into the stage in which they occur; both result in a *fixation*. If a child progresses normally through the stages, resolving each conflict and moving on, then little libido remains invested in each stage of development. But if he fixates at a particular stage, the method of obtaining satisfaction which characterized the stage will dominate and affect his adult personality.

Oral Stage (0 to 18 months)

The oral stage begins at birth, when the oral cavity is the primary focus of libidal energy. The child, of course,

preoccupies himself with nursing, with the pleasure of sucking and accepting things into the mouth. The *oral character* who is frustrated at this stage, is a child whose mother refused to nurse him on demand or who truncated nursing sessions early, and is characterized by pessimism, envy, suspicion and sarcasm. The overindulged oral character, whose nursing urges were always and often excessively satisfied, is optimistic, gullible, and is full of admiration for others around him. The stage culminates in the primary conflict of weaning, which both deprives the child of the sensory pleasures of nursing and of the psychological pleasure of being cared for, mothered, and held. The stage lasts approximately one and one-half years. Problems and tensions in this stage can result in some oral behavior like thumb sucking.

Anal Stage (18 months to 3 years)

At one and one-half years, the child enters the anal stage. With the advent of toilet training comes the child's obsession with the erogenous zone of the anus and with the retention or expulsion of the feces. This represents a classic conflict between the id, which derives pleasure from expulsion of bodily wastes, and the ego and superego, which represent the practical and societal pressures to control the bodily functions. The child meets the conflict between the parent's demands and the child's desires and physical capabilities in one of two ways: Either he puts up a fight or he simply refuses to go. The child who wants to fight takes pleasure in excreting maliciously, perhaps just before or just after being placed on the toilet. If the parents are too lenient and the child manages to derive pleasure and success from this expulsion, it will result in the formation of an *anal expulsive character*. This character is generally messy, disorganized, reckless, careless, and defiant. Conversely, a child may opt to retain feces, thereby spiting his parents while enjoying the pleasurable pressure of the built-up feces on his intestine. If this tactic succeeds and the child is overindulged, he will develop into an *anal retentive character*. This character is neat, precise, orderly, careful, stingy, withholding, obstinate, meticulous, and passive-aggressive. The resolution of the anal stage, proper toilet training, permanently affects the individual propensities to possession and attitudes towards authority. This stage lasts from one and one-half to three years.

Phallic Stage (3 to 5 years)

The phallic stage is the setting for the greatest, most crucial sexual conflict in Freud's model of development. In this stage, the child's erogenous zone is the genital region. As the child becomes more interested in his genitals, and in the genitals of others, conflict arises. The conflict, labeled the *Oedipus complex* (The *Electra complex* in girls), involves the child's unconscious desire to possess the opposite-sexed parent and to eliminate the same-sexed one. The conflict was named by Sigmund Freud after the story of Oedipus rex by Sophocles in the 5th century B.C. In this story, Oedipus, the king unknowingly kills his father, a robber on the highway, and marries his mother, the widow.

In the young male, the Oedipus conflict stems from his natural love for his mother, a love which becomes sexual as his libidinal energy transfers from the anal region to his genitals. Unfortunately for the boy, his father stands in the way of this love. The boy, therefore, feels aggression and envy towards this rival, his father, and also feels fear that the father will strike back at him. As the boy has noticed that women, his mother in particular, have no penises, he is struck by a great fear that his father will remove his penis, too. The anxiety is aggravated by the threats and discipline he incurs when caught masturbating by his parents. This *castration anxiety* outstrips his desire for his mother, so he represses the desire. Moreover, although the boy sees that though he cannot possess his mother, because his father does, he can possess her vicariously by identifying with his father and becoming as much like him as possible: this identification indoctrinates the boy into his appropriate sexual role in life. A lasting trace of the oedipal conflict is the superego, the voice of the father within the boy. By thus resolving this incestuous conundrum, the boy passes into the latency period, a period of libidinal dormancy.

On the Electra complex, Freud was more vague. The complex has its roots in the little girl's discovery that she, along with her mother and all other women, lack the penis which her father and other men possess. Her love for her father then becomes both erotic and envious, as she yearns for a penis of her own. She comes to blame her mother for her perceived castration, and is struck by *penis envy*, the apparent counterpart to the boy's castration anxiety. The resolution of the Electra complex is far less clear-cut than the resolution of the Oedipus

complex is in males; Freud stated that the resolution comes much later and is never truly complete. Just as the boy learned his sexual role by identifying with his father, so the girl learns her role by identifying with her mother in an attempt to possess her father vicariously. At the eventual resolution of the conflict, the girl passes into the latency period, though Freud implies that she always remains slightly fixated at the phallic stage. Fixation at the phallic stage develops a phallic character, who is reckless, resolute, self-assured, and narcissistic—excessively vain and proud.

Latent Stage (6 to 12 years)

The resolution of the phallic stage leads to the latency period, which is not a psychosexual stage of development, but a period in which the sexual drive lies dormant. Freud saw latency as a period of unparalleled repression of sexual desires and erogenous impulses. During the latency period, children pour this repressed libidinal energy into asexual pursuits such as school, athletics, and same-sex friendships. In this stage no organ predominates. But soon puberty strikes and the genitals once again become a central focus of libidinal energy.

Genital Stage (13 to 19 years)

In the genital stage, as the child's energy once again focuses on his genitals, interest turns to heterosexual relationships. The less energy the child has left invested in unresolved psychosexual developments, the greater his capacity will be to develop normal relationships with the opposite sex. If, however, he remains fixated, particularly on the phallic stage, his development will be troubled as he struggles with further repression and defenses.

Psychosocial Theory (Erik Erikson) (Fig. 21.4)

Erikson believed that childhood is very important in personality development. He accepted many of Freud's theories, including the id, ego, and superego, and Freud's theory of infantile sexuality. But Erikson rejected Freud's attempt to describe personality solely on the basis of sexuality, and, unlike Freud, felt that personality continued to develop beyond five years of age. Erikson's psycho-social theory holds certain tenets that differentiate his theory from Freud's. Some of these include:



Fig. 21.4: Erik Erikson (1902-1994)

- The ego is of utmost importance.
- Part of the ego is able to operate independently of the id and the superego.
- The ego is a powerful agent that can adapt to situations, thereby promoting mental health.
- Social and sexual factors both play a role in personality development.

Stages of Psychosocial Development (Fig. 21.5)

Erikson's psychosocial theory essentially states that each person experiences eight 'psychosocial crises' (internal conflicts linked to life's key stages) which help to define his or her growth and personality. People experience these 'psychosocial crisis' stages in a fixed sequence, but timings vary according to people and circumstances. Successful passage through each stage is dependent on striking the right balance between the conflicting extremes rather than entirely focusing on (or being guided towards) the 'ideal' or 'preferable' extreme in each crisis. In this respect, Erikson's theory goes a long way in explaining why too much of anything is not helpful for developing a well-balanced personality.

Development of Basic Trust: Birth to 18 Months (Trust vs Mistrust)

Development of the basic Trust depends on caring and consistent mother or mother substitute, who meets both the physiologic and emotional needs for the infants. The

strong bond between mother and child is necessary for the child to develop a basic trust in the world. The infant will develop a healthy balance between trust and mistrust if fed and cared for and not over-indulged or over-protected. Abuse or neglect or cruelty will destroy trust and foster mistrust. Mistrust increases a person's resistance to risk-exposure and exploration. "Once bitten twice shy" is an apt analogy. On the other hand, if the infant is insulated from all and any feelings of surprise and normality, or unfailingly indulged, this will create a false sense of trust amounting to sensory distortion, in other words, a failure to appreciate reality. Infants who grow up to trust are more able to hope and have faith that 'things will generally be okay'. This crisis stage incorporates Freud's psychosexual oral stage, in which the infant's crucial relationships and experiences are defined by oral matters, notably feeding and relationship with mum. Erikson later shortened 'Basic Trust v Basic Mistrust' to simply Trust vs Mistrust, especially in tables and headings.

Maternal deprivation syndrome: When the child receives inadequate maternal support, he will fail to gain weight and is retarded in both physical and emotional growth. This is seen in children of broken families or who lived in a series of foster homes.

The tight bond between parent and child at the early stage of emotional development is reflected in a strong sense of separation anxiety in the child when separated from the parents. If dental treatment is necessary at an early age it is preferable to do so with the parent present.

Basic mistrust: A child who never developed a sense of basic trust will have difficulty in entering into situations that requires trust and confidence in another person. These individuals are extremely frightened and uncooperative.

Development of Autonomy: 18 Months to 3 Years (Autonomy vs Shame and Doubt)

Autonomy means self-reliance. This is independence of thought, and a basic confidence to think and act for oneself. Shame and doubt mean what they say, and obviously inhibit self-expression and developing one's own ideas, opinions and sense of self. Toilet and potty training is a significant part of this crisis, as in Freud's psychosexual Anal stage, where parental reactions, encouragement and patience play an important role in

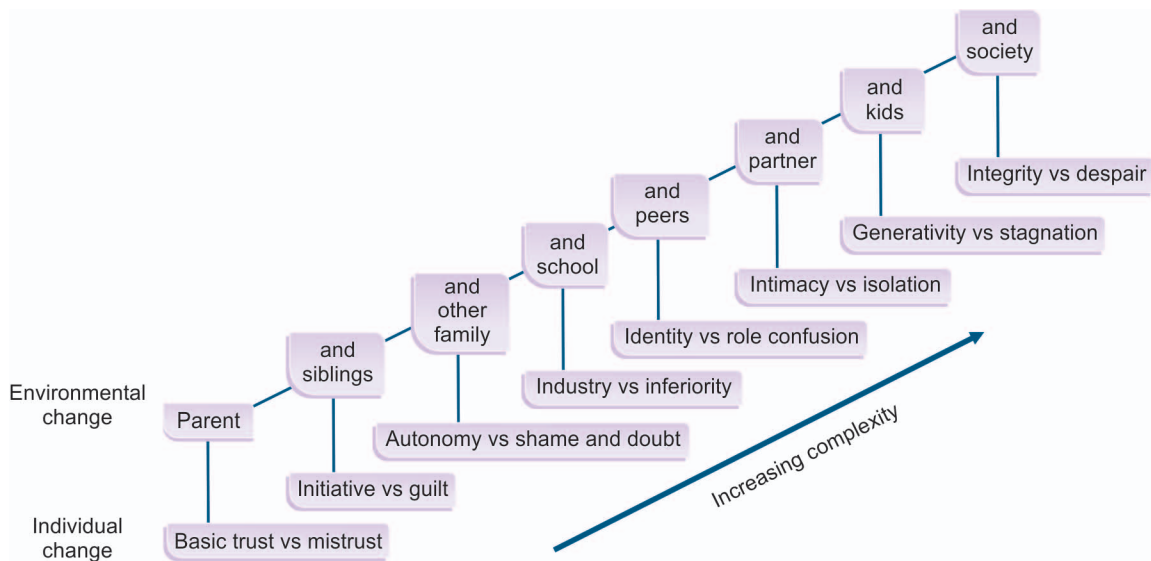


Fig. 21.5: Erikson's stages of development and correlation with social and family environment

shaping the young child's experience and successful progression through this period. Children around the age of 2 years are said to be undergoing *TERRIBLE TWOs* because of their uncooperative nature. At this stage of emotional development, the child is moving away from mother and developing a sense of *AUTONOMY OR IDENTITY*. He varies from a being a little devil to an angel.

Little Devil: He says *NO* to every wish of parents and insists on having his own way.

Little Angel: He retreats to parents in moments of dependence.

Parents and other adults with whom the child reacts at this stage must protect him against the consequences of dangerous and unacceptable behavior, while providing opportunities to develop independent behavior. Consistently enforced limits at this time allow the child to further develop trust in a predictable environment.

Shame and Doubt

Failure to develop a proper sense of autonomy results in the development of doubts in the child mind about his ability to stand alone, and this in turn produce doubts about others. Erickson defines the resulting state as one of shame, a feeling of having one's shortcoming exposed, e.g. bowel control.

This stage is considered decisive in producing the personality characteristic of love as opposed to hate, cooperation as opposed to selfishness and freedom of expression as opposed to self-consciousness. Thus Erickson quotes "From a sense of self control without a loss of self esteem comes a losing sense of goodwill and pride; from a sense of loss of self control and foreign over control comes a lasting propensity for shame and doubt".

A key towards obtaining cooperation with treatment from a child at this stage is to have the child think that whatever the dentist wants was his own choice, not something advised by others. A child who finds the situation is threatening is likely to retreat to mother and be unwilling to separate from her. It is preferable to do dental treatment when one of the parent is present.

Development of Initiative (3-6 years):

Initiative vs Guilt

Initiative is the capability to devise actions or projects, and a confidence and belief that it is okay to do so, even with a risk of failure or making mistakes. Guilt in this context, is the feeling that it is wrong or inappropriate to instigate something of one's own design. During this stage, the child continues to develop greater autonomy, but now adds to it, planning and vigorous pursuit of various activities. For example, extreme curiosity and questioning, aggressive talking, physical activity. This crisis

stage correlates with Freud's psychosexual phallic stage, characterized by a perfectly natural interest in genitals, where babies come from, and as Freud asserted, an attachment to the opposite sex parent, and the murky mysteries of the Oedipus complex.

A major task for parents and teachers at this stage is to channel the activity into manageable tasks, arranging things so that child is able to succeed, and preventing him or her from undertaking tasks where success is not possible.

Guilty: The opposite of initiative is guilt resulting from goals that are contemplated but not attained, from acts initiated but not completed, or from faults or acts rebuked by persons the child respects. Thus Erikson says "The child's ultimate ability to initiate new ideas or activities depends on how well he or she thinks without being made to feel guilty about expressing a bad idea or failing to achieve what was expected".

For most children, the first visit to the dentist comes during the stage of initiative. A child at this stage will be intensely curious about the dentist office and eager to learn about the things found there. So going to the dentist can be constructed as a new and challenging adventure in which child can experience success. Success in coping with the anxiety of visiting the dentist can help develop greater independence and produces a sense of accomplishment.

An exploratory visit with the mother present and with little treatment accomplished, usually, is important in getting a dental experience off to a good start. After this initial experience, a child can tolerate being separated from the mother for treatment and is likely to behave better in this arrangement, so that independence rather than dependence is reinforced.

Mastery of Skills (7-11 years): Industry vs Inferiority

Industry here refers to purposeful or meaningful activity. It's the development of competence and skills, and a confidence to use a 'method', and is a crucial aspect of school years experience. Erikson described this stage as a sort of 'entrance to life'. This correlates with Freud's psychosexual latency stage, when sexual motives and concerns are largely repressed while the young person concentrates on work and skills development. During this period, child is learning about the rules by which

the world is organized and also he is working to acquire the academic and social skills that will allow him to compete in the environment. The influence of parents as a role model decreases and the influence of the peer group increases. Thus Erickson says "The child acquires industriousness and begins the preparation for entrance into the competitive world. However, competition with others within a reward system becomes a reality and also clears that some tasks can be accomplished only by cooperating with the others."

Inferiority: The negative side of emotional development can be acquisition of a sense of inferiority. A child who begins to compete academically, socially, and physically is certain to find that others do something's better. Failure to measure up to the peer group on a broad scale will predispose towards personality characteristic of inadequacy, inferiority, and uselessness.

Children at this age are trying to learn the skills and rules that define success in any situation, that include the dental office. A key to guidance is setting attainable intermediate goals, clearly outlining the child how to achieve this goals and positively reinforcing success in achieving these goals. As the child drives for a sense of industry and accomplishment, cooperation with the treatment can be obtained. Children at this stage are not motivated by abstract concepts. This means emphasizing how the tooth will look better as the child cooperates is more likely to be a motivating factor than emphasizing if you wear the appliance your bite will be better.

Development of Personal Identity (12-17 years): Identity vs Role Confusion

Identity means essentially how a person sees themselves in relation to their world. It's a sense of self or individuality in the context of life and what lies ahead. Role confusion is the negative perspective—an absence of identity—meaning that the person cannot see clearly or at all who they are and how they can relate positively with their environment. This stage coincides with puberty or adolescence, and the reawakening of the sexual urge whose dormancy typically characterizes the previous stage. Adolescence, a period of intense physical development, and is also the stage in psychosocial development in which a unique personality identity is acquired. Adolescence is an extremely complex stage

because of the many new opportunities and challenges thrust upon the teenagers, e.g. emerging sexuality, academic pressures, earning money, increased mobility, career aspirations and recreational interests combines to produce stress and rewards. Establishing ones own identity requires a partial withdrawal from the family, and the peer group increases still further in importance because it offers a sense of continuity of existence in spite of drastic changes within the individual. Thus members of the peer groups become important role models and the values and the tastes of the parents and other authority figures are likely to be rejected.

Confusion: During adolescence, separation from the peer group is necessary to establish ones own uniqueness and values. As adolescence progresses, inability to separate from the group indicates some failure in identity development. This in turn can lead to a poor sense of direction for the future, confusion regarding ones place in society, and low self esteem.

Most orthodontic treatment is carried out during the adolescent years, and emotional and behavioral management of adolescents is extremely difficult. Since parental authority is being rejected, a poor psychological situation is created by orthodontic treatment, if it is being carried out primarily because of the parent needs and not the child. At this stage, orthodontic treatment should be instituted only if the patient feels he/she needs it and not just to satisfy their parents.

Internal motivation for seeking treatment is provided by individuals, own desire for treatment to correct a defect that he perceives in him, not some defects pointed to by authority figures whose values are being rejected away.

Development of Intimacy (Young adult): Intimacy vs Isolation

Intimacy means the process of achieving relationships with family and marital or mating partner(s). Erikson explained this stage also in terms of sexual mutuality—the giving and receiving of physical and emotional connection, support, love, comfort, trust, and all the other elements that we would typically associate with healthy adult relationships conducive to mating and child-rearing. The adult stage of development begins with the attainment of intimate relationships with other individuals. Successful development of intimacy depends on a willingness to compromise and even to sacrifice

to maintain relationships. Other factors that affects the development of an intimate relationship includes all aspects of each person—appearance, personality, emotional qualities, intellect, and others. A significant change in any of the parameter may be perceived by either partner as altering the relationship. Success leads to the establishment of affiliations and partnerships, both with a mate and with others of the same sex in working towards the attainment of career goals.

Isolation conversely means being and feeling excluded from the usual life experiences of dating and mating and mutually loving relationships. This logically is characterized by feelings of loneliness, alienation, social withdrawal or non-participation. Failure leads to isolation from others and set of attitudes than serves to keep others away rather than bringing them onto closer contact.

Most of the young adults seek orthodontic treatment to correct their dental appearance because they perceived their dental appearance as flawed. They may feel that a change in their appearance will facilitate attainment of intimate relationships. On the other hand, a new look resulting from orthodontic treatment may interfere with previously established relationships. Due to of these potential problems, the potential psychologic impact of orthodontic treatment must be fully explained to and explored with the young adult patient before beginning treatment.

Guidance of the Next Generation (Adults): Generativity vs Stagnation

Generativity derives from the word generation, as in parents and children, and specifically, the unconditional giving that characterizes positive parental love and care for their offspring. A major responsibility of a mature adult is the establishment and guidance of the next generation. Becoming a successful parent is not only a major part of this but also services to the group, community and nation. Thus, the next generation is not only nurturing and influencing ones own children but also supporting the network of social services needed to ensure the next generation success.

Stagnation is an extension of intimacy which turns inward in the form of self-interest and self-absorption. It's the disposition that represents feelings of selfishness, self-indulgence, greed, lack of interest in young people and future generations, and the wider world. Stagnation

and/or self-absorption results from not having an outlet or opportunity for contributing to the good or growth of children and others, and potentially to the wider world.

Attainment of Integrity (Late Adult): Integrity vs Despair

This is a review and closing stage. The final stage of psychosocial development is the attainment of integrity. At this stage the individual has adapted to the combination of gratification and disappointment that every adult experiences. Integrity means feeling at peace with oneself and the world. No regrets or recriminations. The feeling of integrity is the feeling that one has made the best of their life.

Despair and/or 'Disgust' represent the opposite disposition: feelings of wasted opportunities, regrets, wishing to be able to turn back the clock and have a second chance. This feeling is often expressed as disguise and unhappiness, frequently accomplished by a fear that death will occur before a life change, that might lead to integrity, can be accomplished.

Conclusion: Erikson's psychosocial theory is very powerful for self-awareness and improvement, and for teaching and helping others. The concept also asserts that humans continue to change and develop throughout their lives, and that personality is not exclusively formed during early childhood years. This is a helpful and optimistic idea, and many believe it is realistic too. It is certainly a view that greatly assists in encouraging oneself and others to see the future as an opportunity for positive change and development, instead of looking back with blame and regret.

Cognitive Theory—Jean Piaget (Fig. 21.6)

The concept of cognitivism was given by Swiss child psychologist Jean Piaget. Piaget rejected the idea that learning was the passive assimilation of given knowledge. Instead, he proposed that learning is a dynamic process comprising successive stages of adaptation to reality. Piaget's theory has two main strands: first, an account of the mechanisms by which cognitive development takes place; and second, an account of the four main stages of cognitive development through which children pass.



Fig. 21.6: Jean Piaget

Mechanism of Cognition Development

The basic principle underlying Piaget's theory is the principle of equilibration: all cognitive development (including both intellectual and affective development) progresses towards increasingly complex and stable levels of organization. According to his concept, childhood development proceeds from an egocentric position through a predictable, step like fashion. The child is an active participant with the environment in the constant incorporation and reorganization of data.

Cognition refers to the higher mental process involved in understanding and dealing with the world around us. Cognition includes processes like perception, thinking, concept formation, abstraction, and problem solving. Basic to all these processes is intelligence. Intelligence is a score derived from an intelligence test indicating how the individual's mental ability compares with that of others of the same development age. Equilibration takes place through a process of adaptation, that is, *assimilation* of new information to existing cognitive structures and the *accommodation* of that information through the formation of new cognitive structures.

Assimilation: is the process of using or transforming the environment so that it can be placed in preexisting cognitive structures. It describes the ability of the child to deal with new situation and problems within his age specific skills.

Accommodation: is the process of changing cognitive structures in order to accept something from the environment. It describes the ability of the child to adapt and change his way of dealing with the world to handle a problem, which at first may be too difficult at his particular age and skill.

An example of assimilation would be the child who has learnt the word 'bird' and will assimilate all flying objects into his or her idea of bird. After accommodation, later when he sees a bee, he accommodates by creating a category of flying objects for bees. Both processes are used simultaneously and alternately throughout life. Through this continuous dual process the child is constantly building various hierarchies of related behavior, which Piaget called *Schemata*. Schemata represents a dynamic process of differentiation and reorganization of knowledge, with the resultant evolution of behavior and cognitive functioning appropriate for the age of the child.

Stages of Cognitive Development (Fig. 21.7)

Piaget delineated four periods of cognition growth, each characterized by distinct type of thinking and in which the child successfully relies more upon internal stimuli and symbolic thought and less upon external stimulation.

Sensorimotor Period (0-2 year)

Characteristics of the sensorimotor stage: The first stage of Piaget's theory lasts from birth to approximately age two and is centered on the infant trying to make sense of the world. During the sensorimotor stage, an infant's knowledge of the world is limited to their sensory perceptions and motor activities. Behaviors are limited to simple motor responses caused by sensory stimuli. Children utilize skills and abilities they were born with, such as looking, sucking, grasping, and listening, to learn more about the environment. A child develops from newborn infants who are almost totally dependent on reflex activities to an individual who can develop new behavior to cope with new situation. During this stage child will develop a rudimentary concept of objects, including the idea that object in the environment are permanent; they do not disappear when the child is not looking at them. The child has little ability to interpret sensory data and a limited ability to project forward or backward in time.

Substages of the sensorimotor stage: The sensorimotor stage can be divided into six separate substages that are characterized by the development of a new skill.

- **Reflexes (0-1 month):** During this substage, the child understands the environment purely through inborn reflexes such as sucking and looking.

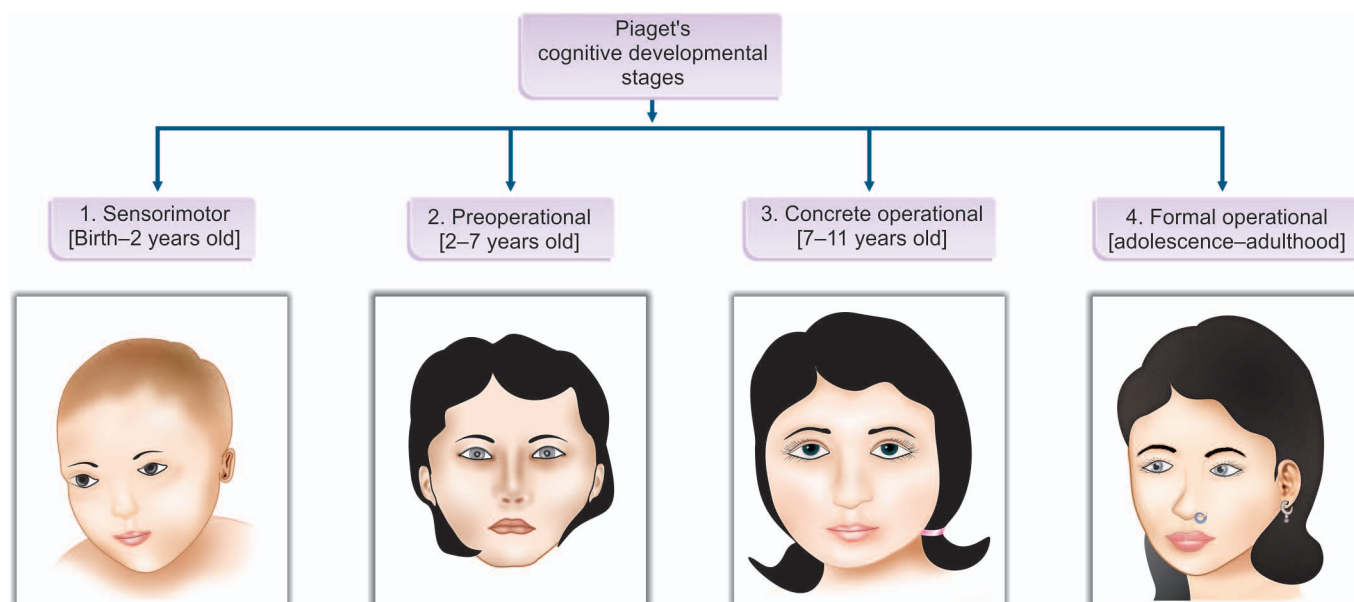


Fig. 21.7: Stages of cognition by Piaget—the four stages are elaborated in text

- *Primary circular reactions (1-4 months)*: This substage involves coordinating sensation and new schemas. For example, a child may suck his or her thumb by accident and then later, intentionally repeat the action. These actions are repeated because the infant finds them pleasurable.
- *Secondary circular reactions (4-8 months)*: During this substage, the child becomes more focused on the world and begins to intentionally repeat an action in order to trigger a response in the environment. For example, a child will purposefully pick up a toy in order to put it in his or her mouth.
- *Coordination of reactions (8-12 months)*: During this substage, the child starts to show clearly intentional actions. The child may also combine schemas in order to achieve a desired effect. Children begin exploring the environment around them and will often imitate the observed behavior of others. The understanding of objects also begins during this time and children begin to recognize certain objects as having specific qualities. For example, a child might realize that a rattle will make a sound when shaken.
- *Tertiary circular reactions (12-18 months)*: Children begin a period of trial-and-error experimentation during the fifth substage. For example, a child may try out different sounds or actions as a way of getting attention from a caregiver.
- *Early representational thought (18-24 months)*: Children begin to develop symbols to represent events or objects in the world in the final sensorimotor sub stage. During this time, children begin to move towards understanding the world through mental operations rather than purely through actions.

The child's knowledge develops in the following areas:

- Object permanence*: Objects continue to exist even when they are not perceived or seen by the child.
- Causality*: Objects have uses and events have causes.
- Symbolic Play*: One object can represent another.

Preoperational Period (2-7 year)

During the preoperational period, the capacity develops to form mental symbols representing things and events not present, and children learn to use words to symbolize these absent objects. Language development is one of the hallmarks of this period. During this period, child can understand the world in the way of five primary

senses, namely touch, smell, hear, taste; concepts that cannot be seen. They feel difficult to interpret time and health.

Features of thought process:

1. Egocentrism
2. Animism
3. Conservation.

Egocentrism: It is defined as the inability of the child to assume another persons point of view. Due to this the child can only manage his own perspective and assuming another's view is simply beyond his mental capabilities. Piaget noted that children in this stage do not yet understand concrete logic, cannot mentally manipulate information, and are unable to take the point of view of other people, which he termed egocentrism.

Animism: It is defined as projection of inanimate object with life, i.e. everything seen as being alive by a young child, and stories that invest with life are quite acceptable to children of this age.

Conservation: Piaget found that few children showed any understanding of conservation prior to the age of five. In one conservation experiment, equal amounts of liquid are poured into two identical containers. The liquid in one container is then poured into a different shaped cup, such as a tall and thin cup, or a short and wide cup. Children are then asked which cup holds the most liquid. Despite seeing that the liquid amounts were equal, children almost always choose the cup that appears fuller.

Most of the thumb sucking patients fall into this category of age. Since the child is egocentric, and he is dominated by how things look, feel, taste, and sound, there is no point in talking to a four year old about how much better his tooth will look in the future if he stops thumb sucking. At the same time, it would not be useful to point out to the child how proud his father would be if he stopped thumb sucking, since the child would think his father's attitude was same as the child (Egocentrism). Telling him that the teeth will feel better now or talking about how bad his thumb tastes will be of use.

Period of Concrete Operations (7-11 year)

During this stage, the ability to see another's point of view develops, while animism declines. The child's thinking is still strongly tied to concrete situations and

the ability to reason on an abstract level is limited. During this time, children gain a better understanding of mental operations. Children begin thinking logically about concrete events, but have difficulty understanding abstract or hypothetical concepts. Presenting ideas as abstract concepts is more difficult to understand than illustrating them with concrete objects. For example, it will be too abstract "Now wear your functional appliance or retainer every night and be sure to keep it clean". More concrete direction would be "This is your retainer. Put it in your mouth like this and take it out like that. Put in every evening right after dinner before you go to bed, and take it out before breakfast every morning. Brush it like this with an old toothbrush to keep it clean".

Features of concrete operations:

1. Logic
2. Reversibility.

Logic: Piaget determined that children in the concrete operational stage were fairly good at the use of inductive logic. Inductive logic involves going from a specific experience to a general principle. On the other hand, children at this age have difficulty using deductive logic, which involves using a general principle to determine the outcome of a specific event.

Reversibility: One of the most important developments in this stage is an understanding of reversibility, or awareness that actions can be reversed. An example of this is being able to reverse the order of relationships between mental categories. For example, a child might be able to recognize that his or her dog is a Labrador, that a Labrador is a dog, and that a dog is an animal.

Period of Formal Operations (11 years - adult)

The formal operational stage begins at approximately age twelve and lasts into adulthood. During this time, people develop the ability to think about abstract concepts. Skills such as logical thought, deductive reasoning, and systematic planning also emerge during this stage. They can understand the concepts like health, disease and preventive treatment. In addition to the ability to deal with abstractions, teenagers have developed cognitively to the point where they can think about thinking.

The important features of this stage are as follows:

1. Imaginary audience
2. Personal fable
3. Logic
4. Abstract thought
5. Problem solving.

Imaginary audience: When an adolescent considers what others are thinking about, he assumes that others are thinking about the same thing he is thinking about, namely himself. They feel they are constantly on stage being observed and criticized by those around them. Elkind has called this phenomenon the *imaginary audience*. The imaginary audience is a powerful influence on young adolescents, making them quite self-conscious and susceptible to peer influence. They are very worried about what peers will think about their appearance and actions, not realizing that others are too busy with themselves.

The reaction of the imaginary audience to braces on his teeth is an important consideration to a teenage patient. They are very susceptible to suggestions from their peer group. In some settings they tend to plead for tooth colored plastic or ceramic brackets and at other times for bright colored ligatures and elastics. When a teenage patient does not want to wear elastics because of peer influence, a useful approach is to agree with him and tell him to try and judge his peer response. It will get him to wear elastics than telling him everybody else does it and he should also to do it.

Personal fable: The adolescent thinks that he is an unique individual, and a second phenomenon emerges which Elkind called the personal fable. The personal fable is a powerful motivator that allows him to cope in a dangerous world. As per this phenomenon, teenagers feel that they are being observed by everybody and this leads them to feel that they are unique individuals. Because of this feeling, they feel that they will not be subjected to dangerous consequences like other people.

Logic: Piaget believed that deductive logic becomes important during the formal operational stage. Deductive logic requires the ability to use a general principle to determine a specific outcome. This type of thinking involves hypothetical situations and is often required in science and mathematics.



Fig. 21.8: Ivan Petrovich Pavlov

Abstract thought: While children tend to think very concretely and specifically in earlier stages, the ability to think about abstract concepts emerges during the formal operational stage. Instead of relying solely on previous experiences, children begin to consider possible outcomes and consequences of actions. This type of thinking is important in long-term planning.

Problem solving: In earlier stages, children used trial-and-error to solve problems. During the formal operational stage, the ability to systematically solve a problem in a logical and methodical way emerges. Children at the formal operational stage of cognitive development are often able to quickly plan an organized approach to solving a problem.

Piaget's work generated interest in child development and had an enormous impact on the future of education and developmental psychology.

Classical Conditioning Theory by Ivan Pavlov (Fig. 21.8)

Pavlov's *Classical Conditioning* was the first model of learning to be studied in psychology. Classical Conditioning investigated the capacity of animals to learn new stimuli and connect them to natural reflexes; allowing non-natural cues to elicit a natural reflex.

Pavlov's experiment (Fig. 21.9): The gist of the experiment is this: Pavlov presented dogs with food, and measured their salivary response (how much they drooled). Then he began ringing a bell just before

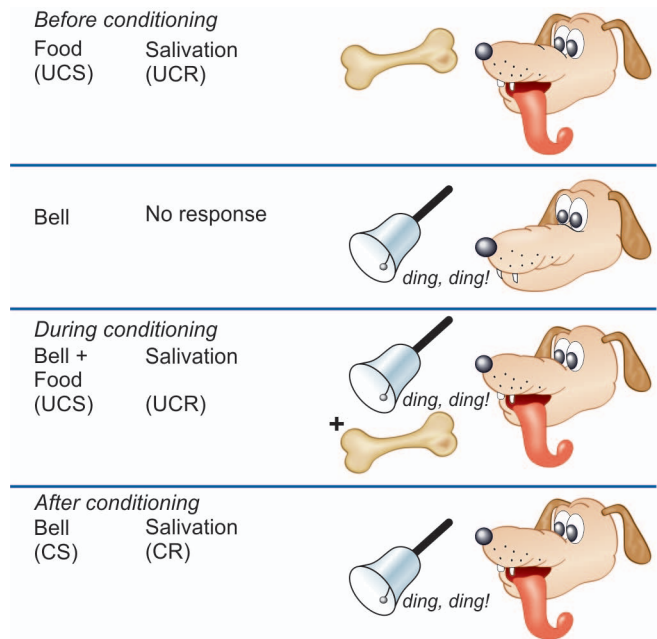


Fig. 21.9: Classic illustration of how unconditioned and conditioned stimuli evoke a response

presenting the food. At first, the dogs did not begin salivating until the food was presented. After a while, however, the dogs began to salivate when the sound of the bell was presented. They learned to associate the sound of the bell with the presentation of the food. As far as their immediate physiological responses were concerned, the sound of the bell became equivalent to the presentation of the food.

Stimuli that animals react to without training are called *primary* or *unconditioned stimuli* (US). They include food, pain, and other "hardwired" or "instinctive" stimuli. Stimuli that animals react to only after learning about them are called *secondary* or *conditioned stimuli* (CS). These are stimuli that have been associated with a primary stimulus. In Pavlov's experiment, the sound of the bell meant nothing to the dogs at first. After the sound was associated with the presentation of food, it became a conditioned stimulus. If a warning buzzer is associated with the shock, the animals will learn to fear it. Thus, Pavlov discovered that an apparently unassociated stimuli could produce reflexive behavior—association of one stimulus with another. Classical conditioning occurs by the process of associating one stimulus with another. Therefore, it is also called "learning by association".

Correlation of conditioned response in Dental practice: Classical conditioning occurs in a dental clinic in the following way:

Initial visit

Hospital atmosphere		Pain of injection
Doctor's coat, nurse's dress	→	Unconditioned stimuli
[Neutral stimulus]		
Pain of injection	→	Fear response and crying [Response]
[Unconditioned stimulus]		

Subsequent visit

Hospital atmosphere	→	Fear Response and crying [Response]
[Doctor's coat, nurse's dress]		
[Neutral stimulus]		

The whole atmosphere of hospital gets associated with pain and the child starts crying the moment he enters the clinic.

Features of Classical Conditioning

Important features of classical conditioning are:

1. Stimulus generalization
2. Stimulus discrimination
3. Extinction
4. Spontaneous recovery.

Stimulus generalization: Pavlov noticed a phenomena he dubbed *stimulus generalization*. If a dog became conditioned to slobber at the sound of a bell, then just about any bell might do. Similarly, if a child experiences pain during every visit to the clinic, then generalization or reinforcement occurs. Child will get a feeling that only painful happenings take place in the clinic. Therefore, painful procedures should be deferred till the end.

Stimulus discrimination: Pavlov also studied *stimulus discrimination*, when the dog would learn that not any bell would do. By this process, the child learns to differentiate between places where painful things do not happen. Continuous discrimination leads to erasing of generalization process.

Extinction: Pavlov found that conditioned responses could be eliminated gradually; a process he referred to as *extinction*. If Pavlov rang his bell repeatedly and failed to feed his dogs, they eventually learnt that the free lunch was over and would once again only salivate in the presence of food. When the association between conditioned and unconditioned stimuli is not reinforced, extinction occurs.

Spontaneous recovery: Pavlov also noticed that extinct conditioned responses could also reappear after a rest period if the conditioned stimulus was again presented some hours later; a process he dubbed *spontaneous recovery*.

Operant Conditioning Theory—BF Skinner (Fig. 21.10)

BF Skinner was one of the most influential of American psychologists. A radical behaviorist, he developed the theory of operant conditioning—the idea that behavior is determined by its consequences, be they reinforcements or punishments, which make it more or less likely that the behavior will occur again.

Theory

Operant conditioning forms an association between behavior and consequence. It is also called *response-stimulus* or RS conditioning because it forms an association between the individual's response [behavior] and the stimulus that follows [consequence] (Fig. 21.11). It is actually an extension of classical conditioning. The organism is in the process of "operating" on the environment, which in ordinary terms means it is bouncing around its world, doing what it does. During this "operating," the organism encounters a special kind of stimulus, called a reinforcing stimulus, or simply a reinforcer. This special stimulus has the effect of increasing the operant—that is, the behavior occurring just before the reinforcer. This is operant conditioning: "the behavior is followed by a consequence, and the nature of the



Fig. 21.10: BF Skinner

consequence modifies the organisms' tendency to repeat the behavior in the future." There are four possible consequences to any behavior. They are: (i) Something good can start or be presented; (ii) Something good can end or be taken away; (iii) Something bad can start or be presented; (iv) Something bad can end or be taken away. Skinner believes that the consequences of a behavior itself can act as a stimulus and can affect future behavior.

Definitions of Terms

Positive: The technical term for "an event started" or "an item presented" is *positive*, since it's something that's *added* to the individual's environment.

Negative: The technical term for "an event ended" or "an item taken away" is *negative*, since it's something that's *subtracted* from the animal's environment.

Reinforcement: Anything that *increases* a behavior—makes it occur more frequently, makes it stronger, or makes it more likely to occur—is termed a *reinforcer*. Often, a person will perceive "Starting Something Good" or "Ending Something Bad" as something worth pursuing and they will repeat the behaviors that seem to cause these consequences. These consequences will increase the behaviors that lead to them, so they are *reinforcers*. These are consequences the person will work to attain, so they strengthen the behavior.

Punishment: Anything that *decreases* a behavior—makes it occur less frequently, makes it weaker, or makes it less likely to occur—is termed a *punisher*. Often, an animal (or person) will perceive "Ending Something Good" or "Starting Something Bad" as something worth avoiding, and they will not repeat the behaviors that seem to cause these consequences. These consequences will decrease the behaviors that lead to them, so they are *punishers*.

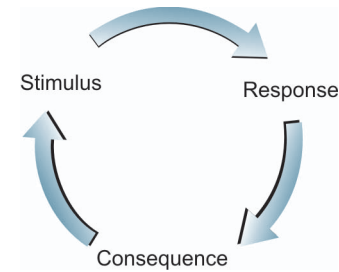


Fig. 21.11: Stimulus, response and consequence feedback loop

Types of Operant Conditioning (Table 21.1)

There are four types of operant conditioning, namely positive reinforcement, negative reinforcement, positive punishment, and negative punishment.

Positive reinforcement: This is possibly the easiest, most effective consequence for a trainer to control. Positive reinforcement means starting or adding something good, something the individual likes or enjoys. Because the individual wants to gain that good thing again, he or she will repeat the behavior that seems to cause that consequence. Reinforcers increase behavior. The reward has to be sufficient to motivate a repetition. The child is likely to behave in the same good manner in subsequent visits also, following positive reinforcement, e.g. child rewarded with toy for good behavior in the dental clinic.

Positive punishment: Positive punishment is something that is applied to reduce a behavior. The term "positive" often confuses people, because in common terms "positive" means something good, upbeat, happy, pleasant, and rewarding. Here "positive" means "added" or "started". Child is likely to behave following this because of the punishment for bad behavior. In clinical practice it should not be used frequently and used only

Table 21.1: Types of operant conditioning

	Reinforcement (Behavior increases)	Punishment (Behavior decreases)
Positive (something added)	Positive Reinforcements Something added increases behavior	Positive punishment Something added decreases behavior
Negative (something removed)	Negative Reinforcement Something removed increases behavior	Negative Punishment Something removed decreases behavior

when other methods fail, e.g. voice control and HOME technique.

Negative reinforcement: Negative reinforcement increases a behavior by ending or taking away something bad or aversive. By making the child's circumstances better, you are rewarding it and increasing the likelihood that it will repeat the behavior that was occurring when you ended the bad thing. In this, the unpleasant stimulus is withdrawn following the behavior of the child. For example, explaining to the child that the treatment procedure is shortened because of the child's behavior.

Negative punishment: Negative punishment is reducing behavior by taking away something good. If the child was enjoying or depending on something good, he/she will co-operate to avoid it getting taken away. They are less likely to repeat a behavior that results in the loss of a good thing. This type of consequence is a little harder to control. Examples will include telling a child that her favorite toy will be taken away or that the child's parents will be sent away.

Social Learning Theory—Albert Bandura (Fig. 21.12)

Observational or social learning or modeling is based primarily on the work of Albert Bandura. Bandura's social learning theory is referred to as observational learning, which implies that new responses are learned through observing the behaviors of others. Rather than experiencing reinforcement for themselves, Bandura argued that people can learn through vicarious reinforcement, which means that we internalize the consequences of other peoples' actions and thus adjust our behaviors as functions of those consequences. Bandura argued that modeling caused disinhibitions, thus the weakening of inhibitions.

According to this theory, all behavior is learnt by reinforcement. The approval of mother acts as a powerful reinforcement of certain emotional development in the child and permits the mother to play an active role to shape and modify the child to socially acceptable level.

Bandura states that excessive emotion is destructive and makes a person acutely uncomfortable. An emotion of a desired limit gives zest of life. Proper emotional development prepares the individual to appreciate the



Fig. 21.12: Albert Bandura

pleasurable aspects of emotion and to cope with unpleasantness in a constructive manner. Positive emotions like affection, joy, and curiosity are helpful and essential to normal development. Negative emotions like fear, anger, and jealousy are harmful to the individual development.

Our families, friends, school, city, and culture are the social background within which we define our own identity and accommodate and assimilate knowledge. It is by interacting with other people and the world that we learn (Fig. 21.13). An observer's behavior can be affected by the positive or negative consequences called vicarious reinforcement or vicarious punishment of a model's behavior. Learning by observation involves four separate processes: *attention*, *retention*, *production* and *motivation*.

Attention: Child cannot learn unless they pay attention to what's happening around them. This process is influenced by characteristics of the model, such as how much one likes or identifies with the model, and by characteristics of the observer, such as the observer's expectations or level of emotional arousal.

Retention: Child must not only recognize the observed behavior but also remember it at some later time. This process depends on the observer's ability to code or structure the information in an easily remembered form or to mentally or physically rehearse the model's actions.

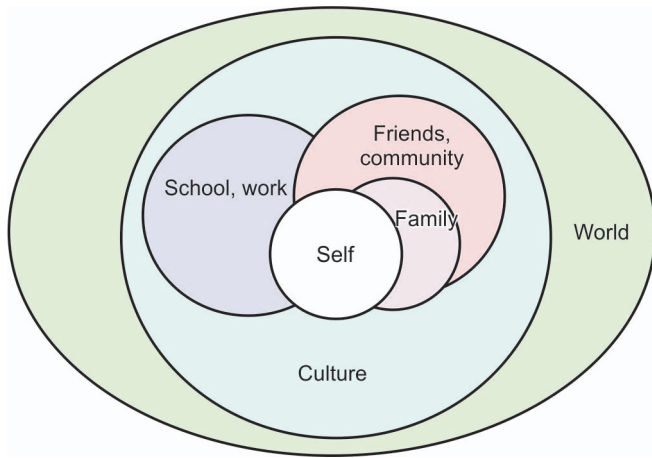


Fig. 21.13: Social learning through interaction with various groups of people

Production: Child must be physically and/intellectually capable of producing the act. In many cases the observer possesses the necessary responses. But sometimes, reproducing the model's actions may involve skills the observer has not yet acquired. It is one thing to carefully watch a circus juggler, but it is quite another to go home and repeat those acts.

Motivation: The fourth subfunction in observational learning concerns motivational processes. People do not perform everything they learn. Performance of observationally learned behavior is influenced by three major types of incentive motivators: direct, vicarious, and self-produced. People are more likely to perform observationally learned behavior if it is rewarding rather than punishing. People are motivated by the successes of others who are similar to them but are discouraged by their failures. People pursue activities that they find satisfying and that confers a sense of self-worth; they reject those of which they personally disapprove.

Observational learning is, thus, coming to play an increasingly influential role in sociopolitical and

transcultural change. In this broader function of social diffusion, modeling through the mass media instructs people in new ideas and social practices. Positive and negative incentives determine which of the modeled innovations will be adopted. Observational learning takes many forms and produces diverse outcomes.

Table 21.2 presents the correlation of various theories of psychology.

Hierarchy of Needs—Abraham Maslow, 1943 (Fig. 21.14)

Maslow's introduction of deficiency needs, B-values and related concepts changed psychology forever. Maslow elevated psychology to a deeper level through the study of great people instead of broken people. Instead of sick-man studies, Maslow did exemplary-man studies. He focused on people such as Albert Einstein, and Eleanor Roosevelt. He cataloged what great values they had in common.

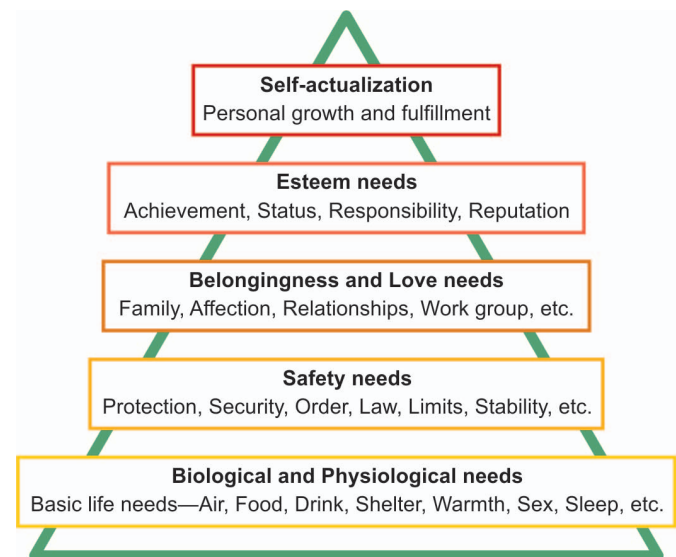


Fig. 21.14: Hierarchy of needs by Maslow

Table 21.2: Correlation of various theories of psychology

Chronological age (years)	Period	Psychoanalytical	Psychosocial	Cognitive
Birth to 1 year	Infancy	Oral stage	Trust	Sensorimotor
1 to 2 years	—	Anal Stage	Autonomy	Preconceptual
2 to 6 years	Early childhood	Phallic stage	Initiative	Preoperational
6 to 12 years	Late childhood	Latent stage	Industry	Concrete operations
12 to 18 years	Adolescence	Genital stage	Identity	Formal operations

Maslow's Hierarchy of Needs is often illustrated as a pyramid of five layers, as each need is based on meeting the needs of the layer beneath it. The four lower levels are grouped together as being associated with physiological needs, while the top level is termed growth needs associated with psychological needs. Deficiency needs must be met first. Once these are met, seeking to satisfy growth needs drives personal growth. The higher needs in this hierarchy only come into focus when the lower needs in the pyramid are satisfied. Once an individual has moved upwards to the next level, needs in the lower level will no longer be prioritized.

Maslow's Basic or Deficiency Needs

The first four layers of the pyramid are called "deficiency needs" or "D-needs": the individual does not feel anything if they are met, but feels anxious if they are not met. The deficiency needs are: Physiological, Safety, Love/Belonging, and Esteem needs.

Physiological needs: Also called as body needs, they include the very basic need for air, warmth, food, sleep, stimulation and activity. People can die due to lack of biological needs and equilibrium (homeostasis). If a person is hungry or thirsty or his body is chemically unbalanced, all of his energies turn toward remedying these deficiencies, and other needs remain inactive.

Safety needs: Also called as security needs, these include living in a safe area away from threats. This level is more likely to be found in children as they have a greater need to feel safe. The individual's safety needs take over and dominate his behavior when the body needs are met. Safety needs include the following:

- Personal security from crime
- Financial security
- Health and well-being
- Safety net against accidents/illness and the adverse impacts.

Social needs: Also called as love or belonging needs. This aspect of Maslow's hierarchy involves emotionally-based relationships like:

- friendship
- sexual intimacy
- having a supportive and communicative family.

Esteem needs: Also called ego needs, these mean having a healthy pride in one's self. The need for self-respect, and respect from others shows up at this level. All humans

have a need to be respected, to have self-esteem, self-respect, and to respect others.

Maslow's Growth Needs

This is the fifth layer in the triangle. Also called as fulfillment or self actualization needs, these include purpose, personal growth and realization of one's potentials. This is the point where people start to become fully functional, acting purely on their own volition and having a healthy personality. Self-actualization is reaching one's fullest potential.

Psycho-orthodontic Theory

Psycho-orthodontic theory of motivation was put forwarded by El-Mangoury. Motivation is a very broad psychological term which describes a hypothetical construct which aims to explain the reason for the stream of a goal-directed behavior driven by specific or nonspecific forces. There are three types of motivation:

- i. *Achievement motivation* can be defined as the motivation characterized by striving for success in any situation in which standards of excellence apply.
- ii. *Affiliation motivation* of orthodontic patients is defined as a hypothetical construct of seeking orthodontic care for the purpose of improving the dentofacial esthetics in order to facilitate the connection or association of oneself with other people for obtaining, maintaining, and/or restoring close interpersonal relationships.
- iii. *Attribution motivation* can be defined as the motivation for perceiving the causes of success and failure, either internally (that is, to the self) or externally (that is, outside the self).

El-Mangoury did a detailed psychological study among orthodontic patients. On the basis of the results obtained from this study, the following conclusions were drawn:

1. Orthodontic cooperation is predictable through psychological testing.
2. High-need achievers cooperate better orthodontically than low-need achievers.
3. High-need affiliators cooperate better orthodontically than low-need affiliators.
4. Internals cooperate better orthodontically than externals.
5. Orthodontic cooperation does not involve a simple single general dimension of cooperation.

6. Orthodontic cooperation is composed of a more complex structure of two separate orthogonal constructs: the specific orthodontic construct of cooperation (SOCC) and the perio-orthodontic construct of cooperation (POCC).
7. As SOCC and POCC are orthogonal (that is, unrelated), a patient who is a good brusher does not have to be a good headgear wearer, and vice versa.
8. The single best summary of the linear relationship exhibited by orthodontic cooperation is SOCC. The second best summary is POCC.
9. Affiliation motivation seems to contribute the most to the prediction of headgear wear, elastic wear, appliance maintenance, nonbroken appointments, and punctuality in appointments.
10. Achievement motivation appears to contribute the most to predicting oral hygiene.

HABIT INTERVENTION AND EMOTIONAL GROWTH

Graber defined habits as the tendency towards an act that has become a repeated performance, relatively fixed, consistent and easy to perform by an individual.

Thumb Sucking

Of all the oral habits, thumb sucking is probably the one that merits most discussion. The recommended procedures extend from very early treatment, to treatment at an older age, to no treatment at all. Unless the complexities of the problem are understood, efforts at correction are likely to become exercises in frustration. Two main schools of thought prevail.

The psychoanalysts regard the habit as a symptom of emotional disturbance. The psychoanalysts believe that sucking in infancy (birth to 2 years) is part of the normal behavior pattern, which satisfies two needs, that of taking food and that of oral gratification. Very frequently the nutritional requirements of infants are amply catered to, but the actual "sucking need" has not been satisfied. This could be caused by feeding bottles with large apertures, causing the child to gulp the food rather than working for it by the sucking action. Having not satisfied the emotional needs, the infant fulfills the sucking requirement with the readily available fingers or thumb. The sucking needs vary from two hours in some infants to only a few minutes in others. Sucking reaches its maximum intensity at four months and tends

to wane at different ages in different individuals, usually toward the latter half of the first year. Therefore, to wean a child abruptly or to change from a liquid to a solid diet before the age of 4 months may cause the child to suck on any object, usually a digit, to satisfy the emotional need. The difference in incidence of persistent thumb sucking in infants reared by bottle or breast is not significant.

The *behaviorists* view the act as a simple learned habit with no underlying neurosis.

Oral drive theory (Sears and Wise): Sears and Wise state that thumb sucking is not because of weaning of feeding.

- Thumb sucking is the result of the prolonged drive for suckling or nursing.
- They further stated that sucking increases the erratogenesis of the mouth.

Benjamin theory: This theory attributes thumb sucking to two reasons:

1. Thumb sucking is an expression of the need to suck because of the association with reinforcing aspect of suckling or feeding.
2. Another reason for thumb sucking is that it is because of the rooting and placing reflexes common to all mammals. During infancy, the child will have a tendency to place the objects in the mouth. It is maximum during 3 months of age and gradually disappears by 7 to 8 months.

Learning theory: states that thumb sucking is associated with unrestricted or prolonged nutritive sucking.

Preschool Child

In the preschool child (2 to 5 years) mild sucking before retiring from any work or when fatigued is normal. In most instances, children who indulge in the habit at this age are those who have continued to do so from infancy. In instances where the habit is initiated in the preschool years, the cause is generally emotional. Such habits may appear during a period of sibling rivalry or when the child feels that the interest of the parents is being absorbed elsewhere. Sucking at this age may appear during times of stress, which is a regression to an earlier pleasure and sense of security associated with suckling at the breast when mother and baby were a biologic unit. Damage to tooth position is dependent upon duration, frequency, and intensity of the habit. Temporary malpositions of the deciduous teeth may

result from continuous vigorous thumb- or finger-sucking. If the habit is discontinued before the sixth year, the deformity will be corrected spontaneously in about 75 percent of cases, provided again that the lip musculature is competent.

School Child

Thumb sucking in the schoolchild (6 to 12 years) is usually a manifestation of a general emotional and social immaturity. Most thumb-suckers in this age group have continued these habit patterns from infancy. As before, the effect on the dentition is dependent upon the intensity, frequency, and duration of the act. Not all thumb-suckers develop malocclusions; nor do all patients with malocclusions have a history of thumb sucking.

Treatment Approach

In treating habits in this age group, it is necessary to determine whether the habit is "meaningful" or "empty." If the sucking habit is one of a galaxy of symptoms of an abnormal behavior problem, a consultation with a psychiatrist is the first consideration. The habit in these instances would be regarded as "meaningful." "Empty" habits frequently are broken by simply discussing them with the patient. It may be difficult to assess the nature of the habit, in which case the general treatment plan is aimed at correction of the habit in a manner, which will not result in psychological trauma to the child.

Psychological Approach

The simplest approach to habit therapy is a discussion between the child and dentist.

- This is most effective in older children.
- No threats or shaming should be used.
- A calm and friendly attempt should be made to educate the child about the ill effects of the habit.
- Child may be shown photographs or study casts of children who had deleterious sucking habits.
- Cards can be given to children for scoring each morning to indicate whether the thumb was sucked during night. This produces good result.

Dunlap's beta hypothesis: Dunlap put forward the Beta hypothesis.

- According to this theory, conscious purposeful repetition is the best way to discontinue a habit.

- The child is made to sit in front of a large mirror and asked to suck his finger, observing himself in the mirror.
- This procedure has proved effective in many children.

Audio visual aids: are used for explaining the effects of thumb sucking.

Bruxism and TMJ

James in Angle 1992 showed bruxism is considered as one predisposing sign of myofascial pain dysfunction (MPD) syndrome which is often thought to result from multiple causative factors. These causative factors may include psychologic, emotional, dental, systemic, occupational and/or idiopathic elements. The effects of bruxism are multiple and diverse and include temporomandibular joint pain and dysfunction, head and neck pain, muscle pain and spasms, tooth wear, mobility and damage to supporting structures.

Susan in Angle 1994 showed there is lack of significant association between dentofacial morphology and bruxism and also implied that the etiology of bruxism may not be "structurally" related. By a process of elimination, this would lend credence to the hypothesis that bruxism is of emotional origin and/or a central nervous system phenomenon, rather than due to "form".

McLaughlin in 1988 showed that Schwartz proposed the psycho-physiologic theory of TMJ dysfunction. According to their theory, emotional stress played a much greater role in the etiology of TMJ dysfunction than did "dental irritants."

THE ROLE OF MALOCCLUSION IN PSYCHOLOGICAL DEVELOPMENT

The appearance of the teeth in the mouth and smile plays an important role in judgment of facial attractiveness. Children of normal dental appearance are judged to be better-looking, socially interactive, more desirable as friends, and more intelligent. The teeth have been reported to be the fourth most common teasing target after height, weight and hair (Shaw, 1980). Children have reported that the appearance of their teeth is a common target of teasing. In particular, malocclusions in the anterior region are the most conspicuous and raise the child's greatest concerns. Shaw also found that an overjet of 7 mm or more, anterior crowding and deep

bite are associated with a child's report of being teased. Overjet has also been found to be the most significant predictor of the decision to seek orthodontic correction, especially in children referred for treatment by their parents. Helm, 1985, has found that overjet, extreme deep bite and crowding are associated with the most unfavorable self-perceptions of teeth. Wheeler and Keeling, 1994, showed the demand, or self-perception of need, for orthodontic treatment is greater in female subjects than in male subjects, white subjects than black subjects, urban settings than rural settings, and among children of higher socioeconomic status.

Heldt, Haffke and Davis in 1982 showed that patients with dentofacial deformities, regardless of severity, are frequently the victims of ridicule, teasing, and jokes. The emotional trauma is evident in interviews with patients victimized by this abuse. Dentofacial defects are extremely prominent and, unlike other physical handicaps, cannot be easily disguised. The reactions of 10- and 11-year-old children (representing a variety of geographic locations, races, and cultures) to six pictures of children with various handicaps were studied by Richardson. The six pictures included a child with no physical handicap, a child with crutches and a brace on one leg, one child in a wheelchair, a child with one hand missing, a child with a facial deformity, and an obese child. Almost universally, when asked to rank from most to least pleasing in appearance, the child with the facial deformity was ranked below all except the obese child.

Perkin and Lerner in 1995 found the facial attractiveness ratings by self and others are the best predictors of psychological functioning in adolescents. Thus a child with good facial appearance receives more favorable competence and behavior rating by his teacher than less attractive child. So, attractive children have a built in advantage as they interact with the world outside their nuclear family. They are given more attention and help in learning new skills than less attractive children. However, this relationship holds only for children, not for adults. As they mature, they must show real skills and knowledge that are gained through their own initiative, regardless of the help they have or have not received from others.

Alices Tung in 1998 showed that a teacher's perceptions of a child's attractiveness can influence the teacher's expectations and evaluation of the child. Children perceived as more attractive are not only more

socially accepted by their peers, they are also believed to be more intelligent and to possess better social skills. In addition, people perceived as attractive by their peers are considered more desirable as friends than are unattractive people.

Self Concepts

Self-concept is defined as the perception of one's own ability to master or deal effectively with the environment. The individual's interactions with and responses from others may influence the development of self-concept. Developmental psychologists generally agree that a child's self-concept develops from the 'reflected appraisal' that he or she receives from others. Thus, self-concept is affected by the reactions of others toward the child. Self-concept also depends on social comparisons and self-attributions by the child. As discussed earlier, facial attractiveness plays an important role in social acceptance by peers. A positive relationship also exists between physical/facial attractiveness and interpersonal popularity, as well as others' favorable evaluations of personality, social behaviors, and intellectual expression.

Personality theories emphasized the importance of physical appearances in self-concepts. However, physical appearances are not the only factor that determines self-concepts and self-esteem. Other factors like academics, athletic achievements, ability of interaction with the peers, teachers, and others all come to play an increasingly important role in self-concepts.

Teeth vs Facial Attractiveness

Berscheid and Walster, 1973, found that the face was the most important physical characteristic in the development of high self-esteem (male and female); that is, persons who are satisfied with their faces are more self-confident.

It was also cited that both men and women expressed more dissatisfaction with their teeth than with any other facial feature.

Females vs Males

Females have consistently been found to have more negative body image and self-concept scores. This phenomenon begins in adolescence, when girls become more concerned about their physical appearance and weight. Although pubertal changes increase the self-

consciousness of boys and girls, the latter are more influenced by these rapid changes in their physical appearance, and they continue to attach more importance to these external characteristics into adulthood. Thus, girls in particular, express greater concern about their facial features, especially when certain features (teeth, nose, hair) are different from those of their peers.

Parental Status

Parental concern most likely stems from the parent's hope that the child will conform to their own and society's ideals of facial attractiveness. It has been suggested that parental influence based on dental esthetics, not necessarily malocclusion severity, may be the main motivating factor for children to seek orthodontic treatment. Thus, the degree of malocclusion does not affect the decision to undergo treatment as much as the perceived esthetics of the malocclusion.

Although overall self-concept has not been found to be altered by orthodontic treatment, some components of self-concept, perceptions of appearance by others (e.g., parents and peers), and body image have been found to improve after treatment. In children with more conspicuous facial impairments such as cleft lip or palate, correction may result in improved school performance and social acceptance.

Lerner in 1989, found that self-esteem is the child's internalization of others judgment of his or her attractiveness. However in adolescence, it is a subjective assessment of his or her physical attractiveness, not with objective appraisals by teachers and peers. Thus, children who underrate their own facial attractiveness have been found to score lower on measures of self-esteem than children who rate themselves at or above other's ratings.

Emotional Development and Orthodontic Treatment Need

Body Image

Body image of the patient is classified into "body sense" and "body concept." Body sense refers to the actual appearance the person sees when viewing him in a mirror or photograph. Body concept is the internal process of how the patient feels about his appearance. Body concept is affected by parents, peers, teachers, culture, and also by ethnics.

Parent and Peers

The earliest influences on a child's body awareness are a parent or other caregiver's physical and emotional interaction with the child. As the child's world expands, teachers and peers respond to his or her physical appearance. These messages may reinforce each other and the child's subjective assessment may conflict the child's own perceptions. By integrating these appraisals (and in some cases by ignoring objective judgments) the child develops a cognitive representation of the self, a body image.

Culture and Ethnics

A person's response to dental-facial attractiveness can be viewed as a type of psychosocial response to occlusal status. As such, psychosocial responses to dental-facial esthetics have a cultural emphasis. It is important to assess objectively the degree to which a person's dental-facial appearance deviates from the cultural norm. Thus, there is a rational and empirical basis for including an assessment of dental-facial appearance when evaluating the need for orthodontic treatment. Thus, ethnic and cross-culture factors play a role in the development of a body image.

Self-concept and Self-esteem

Although body image has been represented as an important component of self-concept (or self-identity), it is not the sole factor. Especially, as the child reaches adolescence, his or her accomplishments in academics and athletics, as well as social competence (e.g. ability to play well with peers, showing appropriate class room behaviors) have a significant impact on other's reactions to the child. These responses from others in turn influence the child's self-concept and self-esteem (i.e. ones assessment of self-worth).

To the extent that the child holds himself or herself in high regard, there is greater self-acceptance and the desire to maintain the status quo. For such children, an orthodontist's recommendations or a parents encouragement to obtain orthodontic treatment may be futile because the child is satisfied with his or her appearance, no matter how far outside the range of "ideal" or even normal his dentofacial features may lie. In such cases, if the child is forced by the parents to receive treatment, cooperation during active treatment

and adherence to long term treatment recommendations may suffer.

In contrast, for many children whose self-acceptance is not very high, the desire to change one or more components of self-concept may be great. Those who can identify the malocclusion or poor dentofacial disharmony as the source of their dissatisfaction are more highly motivated to obtain orthodontic treatment and are better risks for long-term cooperation and adherence to treatment protocol.

It behooves the orthodontist to recognize these differences, to identify children who attend the initial orthodontic consultation willingly versus those who are coerced by parents or other concerned adults, as well as those whose own parents motives are unrealistic and inconsistent with the type of malocclusion presented. This requires an honest discussion with the child, perhaps with the parent listening but not participating in the session. Questioning the child about his or her areas of satisfaction with the face and other aspects of the self, motives for and concerns about treatment, and whether or not the child understands his or her responsibilities during each phase of treatment can prevent failure in the case of children who are unprepared or, more importantly, those who have few intrinsic motives for seeking orthodontic intervention.

Treatment During Preadolescence or Adolescence

The decision of whether to treat a patient in childhood or adolescence raises several issues related to the developmental stages of preadolescence and adolescence. One of these issues is the concern with adherence. Treatment adherence is influenced by a child's sex and age. In general, girls are more likely to adhere to treatment recommendations than boys. Preadolescent children have been found to be more adherent to rules for the use of removable appliances than adolescents. For this reason, it has been suggested that treatment begin after age 6 and be completed before the onset of puberty. Other predictors of greater adherence include high self-esteem, optimism regarding the future, and low social alienation.

Children experience major changes in these aspects of the self as they move from early childhood through the teen years. According to Erikson's theory of psychosocial development, the preadolescent

experiences the stage of "industry vs. inferiority". When social and academic skills develop, children begin to compare their capabilities in these areas with peers, and they increasingly recognize that they can achieve competence through their own initiative. The adolescent goes through a period of "identity vs. role confusion," Erikson's fifth stage of psychosocial development. This is a period of role confusion for many adolescents as their physical selves mature into their future adult selves yet they are still treated as children. The goal of this developmental stage is the search for identity, or "a feeling of being at home in one's body, a sense of knowing where one is going, and an inner assuredness of anticipated recognition from those who count.

Adolescence is often associated with increased self-consciousness, confusion about identity and acceptance by others, and concerns about recognition from adults and peers. Younger children are influenced greatly by their parents and other adults (e.g., teachers, health care providers). As the child enters adolescence, however, peers assume a greater role in their lives, especially in terms of self-image. Peers often serve as a standard of comparison and implicit or explicit critics of the adolescent's appearance, dress, activities, and interests. The ambiguity and fluidity of these peer relationships and the reliance on peer acceptance and ambivalence about parental authority can lead to social alienation but can also provide adolescents with important challenges that help them achieve a sense of identity or "inner assuredness". Indeed, the social, emotional and often academic crises of adolescence are viewed by some personality theorists as a healthy process of reconstructing one's identity and self-concept. Other developmental psychologists have found that self-concept does undergo some change during adolescence but that these changes are not necessarily traumatic.

The increasing significance of peer acceptance for adolescents results in greater need for social comparison. Girls, in particular, express greater concern about their facial features, especially when certain features (teeth, nose, and hair) are different from those of their peers. Boys are not immune to the social-comparison process, but they are more likely to express concerns with their athletic ability and physical size compared with their peers. This increased focus on the self, related to his or her peers may help or hinder the child's success with orthodontic interventions. If the adolescent has significant

concerns about the appearance of his or her teeth and has friends who are undergoing or have undergone orthodontics, they can serve as role models for the child. This role-modeling can result in greater cooperation with the treatment regimen. If, however, the child is absorbed in other developmental tasks of adolescence, it may be the wrong time to initiate treatment. It is evident that adolescents in this study focused most on their past selves, least on their future. Yet they were more likely than the younger subjects to perceive changes in themselves since early childhood (e.g., "Since middle school, I've changed a lot in my personality"). In contrast, the 6 and 9-year olds were more likely than adolescents to think of their future selves (e.g., "I hope someday I'll become an artist") and to view themselves as having experienced few changes in their lives and in their personalities so far. These differences may have implications for children's attitudes toward, and adherence to, orthodontic treatment. Adolescents focused on the "here and now" may have more difficulty with long-term adherence in the interests of future improvements in their oral function and appearance.

EMOTIONAL DEVELOPMENT AND ITS RELATION TO COOPERATION IN TREATMENT

Patients usually expect improved dental facial appearance as an outcome of the treatment, but factors like cooperation play a major role. Nanda, in 1992, showed that female adolescent patients showed more cooperation than male patients. The problem of cooperation of adolescence is mainly due to the social and developmental issues. The establishment of personal values and goals is the salient focus of the teenagers and the influence of parents vary from promoting adaptive behavior to providing standards against which to rebel. Thus the relative strength of peers and parental influences are changing during adolescence maturation.

Gross in 1985 reported adolescents have negative perception of orthodontic treatment and parental support is critical to treatment success.

Kegeles, 1990, reported that children whose parents encouraged treatment were generally cooperative. Cooperation was still higher for the adolescent patient whose parents express positive attitudes towards orthodontic treatment. Kreit found uncooperative patient typically had poor relationship with parents.

In contradiction to the above statement:

1. In University of Buffalo Studies, it was speculated that parental influence declines as children move into adolescence, but no relationship between age of patient and cooperation in treatment was found.
2. Tung and Kiyak 1998 suggested that preadolescence group is the ideal candidate for the treatment because they are not dealing with the issue of identity confusion and concern more about acceptance of others.

Compliance is "the extent to which a person's behavior (in terms of taking medications, following diets, or executing lifestyle changes) coincides with medical or health advice." Compliance of the patient depends upon the motivation and psychological drive of the patient. According to Wick Alexander, there are three requirements for creating patient compliance: belief in one's technique, communication with the patient and motivation. Internal motivation which is provided by the individual's own desire for treatment to correct the defect is the best form of motivation. External motivation due to pressure from friends also will be effective during adolescence.

The complexities of human life and development being as numerous, it would seem, as the stars in the sky, it is no wonder that a single definitive science of human development and behavior has not been, and perhaps cannot be established. However a knowledge of emotional growth helps in studying the patients better and also in dealing with them. It is frequently necessary to initiate treatment early in children who are particularly self-conscious, timid, or sensitive about their dental appearance, even if the dentoskeletal morphology is such that treatment could safely be deferred until later, say, in the late mixed dentition. On the other hand, it might be as necessary to defer treatment in patients who are physiologically ready for it but are emotionally immature or not willing to cooperate at that age. Treatment may have to be delayed even at the expense of losing out on the advantages of growth. Patient's cooperation is vital to achievement of excellent results; without it, treatment becomes a futile exercise.

Thus, the orthodontist become duty bound to carefully evaluate emotional development of the child and adapt his language so that concepts are presented in a way that the patients can understand better. It also helps to understand the patient properly and their needs and help the orthodontist to treat effectively and efficiently.

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